



KASR EL - AINI

INTRODUCTION TO SURGERY

SEVENTH EDITION
NEW EDITION

VOLUME 1

DEDICATION

To those who preceded us and are no longer with us
To those who preceded us and are still among us
And to those who will follow us
We dedicate this book

PREFACE

As is clear from the dedication of this book, the driving feeling behind this effort has been a sense of belonging to this chain-like continuum of generations that our hospital Kasr El-Aini has seen passing within its walls, each generation giving way to a new one, but not before producing an indelible mark of its own, unique to it and forever influencing subsequent generations.

Each year sees a few hundred young men and women treading their first steps in medical learning while at the same time a few hundred others will leave this venerable institution armed with the knowledge they have acquired within its walls and with which they are going to start their journey along the arduous path of medical practice.

To us teachers the most enjoyable and certainly the most fascinating aspect of our profession is the actual moulding of minds and personalities that we help to bring about between the start and the end of medical learning. This process has been likened, and not unjustly, to the moulding of a piece of clay by the hands of a sculptor into a coherent and well balanced piece of statuary. It is not therefore surprising that as soon as the idea of this book was formulated, we all jumped with alacrity at this opportunity of expanding the knowledge and directing the thoughts of our students along the proper path.

In this book each of us has tried in his own field and in his own way to present the subject in question in a way that will help the student acquire easy and readily accessible knowledge and will also help him to grasp the unity of medical knowledge, a thing so vital to a deep understanding of the significance of surgery.

In more practical terms much emphasis has been laid on physiology, anatomy and pathology thus never presenting surgery as an isolated science. The clinical picture of each disease has been discussed at great length and its relation to abnormal physiology and anatomy made quite clear while the details of sophisticated operative procedures have been omitted. Again and adhering to the same philosophy we have discussed accident and emergency surgery at great length seeing that it is these problems that the young surgeon will probably have to face early in his career. All the recent advances in the surgical and para-surgical sciences have been included in the text in a clear and concise manner.

In short we have tried to produce a textbook of surgery that medical students as well as young surgeons will find useful.

We thus present our work hoping that those who preceded us will look upon it with approval and that those who will follow us will carry on after us and dedicate newer versions of this book to us who will have preceded them.

PREFACE TO THE THIRD EDITION

The first two editions of Kasr El-Aini Introduction to Surgery were favourably received by both students and colleagues. Meanwhile, we kept open for their criticism and suggestions. We felt that they are sincere in their desire to develop the book that carries the name of our medical school.

Accordingly it was decided that the theme of this new production would be "a student-friendly edition". The content was both updated and trimmed down to suit what an undergraduate needs to know about surgery. We also focused on making knowledge easily picked up by providing more figures, tables, and reminder boxes. A new page format was introduced to serve this purpose.

In this edition the anaesthesia chapter has been removed. Our colleagues in anaesthesia department have produced their own book "Anesthesia for Medical Students". We strongly recommend this book as a companion to Kasr El-Aini Introduction to Surgery.

We hope that the third edition will be more useful and enjoyable to our students. Their comments and suggestions will always be considered for future refinement of the book.

The editors

PREFACE TO THE FIFTH EDITION

The first editions of this surgery manual were met by enthusiasm from students and from faculty members. We have also received some useful criticism, and plenty of useful ideas and suggestions.

For these reasons we were prompted to produce this fifth edition. Most of the chapters have changed, some of them minimally, while a few were completely revised. Moreover, a new chapter about "minor procedures" was added. In this edition the changes included addition of recent advances, deletion of outdated and redundant knowledge, and trimming of some operative details that are not relevant to the undergraduate curriculum. Above all, the focus was on making this edition more reader-friendly. In this context we hope that it will allow for easy understanding, easy recall of knowledge, and wiser application in a clinical setting.

We believe that the fifth edition of Kasr El-Aini Introduction to Surgery is a useful book for the undergraduate medical student, as well as for the young surgeon in training. Furthermore, it provides the essential knowledge that is needed for surgeons who seek higher qualifications.

We wish to express our gratitude to colleagues from neurosurgery, orthopaedic surgery, urology and anaesthesia who participated generously by writing the corresponding chapters.

We wish to express our great appreciation to the Ciba Geigi Pharmaceutical Company represented by the Ciba Scientific Office, Cairo, Egypt for providing us with plenty of very illustrative figures which are of considerable value. All the X-ray pictures were provided by the surgical staff.

We are deeply indebted to all the staff of Al-Ahram press who did their best to bring out this book in an elegant form.

We are very thankful to Mr. Sayed Mahmoud, the director of the University Book Center, for publishing this book.

Finally we would like to thank our students for their enthusiasm and encouragement.

PREFACE TO THE SEVENTH EDITION

The undergraduate medical student of today is in a difficult situation. This is because the evolution of surgical science has become too fast to follow. Furthermore, the practicing surgeon is required to adopt a practice that is based on the best available medical evidence, a duty that requires exhaustive research, sometimes in situations that require a fast precise decision making.

We, at the department of general surgery of Cairo University still believe that a regularly- updated good textbook is a great help to student and to practicing surgeon alike. Though a heavy task, we decided to accept the challenge and to update this book. The success of previous editions, the encouragement of our colleagues in the profession and the unlimited support we got from our chairmen prompted us to do it.

In this edition old knowledge has been removed and all chapters were updated according to the principles of "evidence-based medicine. Some chapters have been expanded to encompass the tremendous advances made in interventional radiology, oncology, laparoscopic surgery, bariatric surgery and molecular biology.

In addition, and in order to prepare our students and junior surgeons to meet the realities of modern practice, we have added two new chapters; pre and postoperative management of the surgical patients and principles of modern surgical practice.

We would sincerely like to thank our students for their useful comments and suggestions and thank all contributors for generously sharing their knowledge and experience.

CONTENTS

Volume I

1. Wound healing and management	1
2. Major trauma and the multiple injury patient	10
3. Fluid, electrolyte and acid-base balance	17
4. Acute haemorrhage and blood transfusion	30
5. Haemostasis	40
6. Shock	48
7. Surgical infections	60
8. Burns and principles of plastic surgery	86
9. Surgical nutrition	98
10. Tumours	105
11. Organ transplantation	118
12. Skin and subcutaneous tissue	125
13. Arterial disorders	139
14. Venous disorders	172
15. Lymphatic system	190
16. Muscles, tendons and fascia	206
17. Hands and feet	214
18. Surgery of nerves	224
19. Scalp, skull and brain	243
20. Face, lips and palate	266
21. Mouth, cheek and tongue	275
22. Teeth, gums and jaws	282
23. Salivary glands	290
24. The neck	300
25. Pharynx and larynx	311
26. Endocrine surgery	317
27. Breast	356
28. Cardiothoracic surgery	383
29. Oesophagus	412
30. Stomach and duodenum	428

CONTENTS

Volume II

31. The liver	458
32. Biliary system	490
33. Pancreas	515
34. Spleen	531
35. Peritoneum, omentum and mesentery	545
36. Intestine	559
37. Paediatric surgery	605
38. Appendix	619
39. Anal canal	630
40. Review subjects	648
41. Abdominal wall and hernias	664
42. Diagnosis of urinary tract diseases	694
43. Urology; congenital lesions	706
44. Urinary tract injuries	715
45. Urinary tract infections	725
46. Obstructive uropathy	736
47. Urinary tract tumours	762
48. Testes and scrotum	776
49. Fractures, general principles	795
50. Fractures and dislocations of upper limbs	808
51. Fractures and dislocations of the pelvis and lower limbs	822
52. Infections of bones and joints	840
53. Generalized disorders of bones and joints	851
54. Bone tumours	856
55. Deformities of bones and joints	871
56. Amputations	878
57. Surgery of the spine and spinal cord	884
58. Minor procedures	900
59. Pre and postoperative management of the surgical patients.	905
60. Principles of modern surgical practice	910

CONTRIBUTORS

(Arranged alphabetically)

1. ABDEL FATAH NADA
2. ABDEL KADER KOTB
3. ADEL F. RAMZY
4. ADEL HOSNY
5. AHMED ADEL
6. AHMED EL-BISHRY
7. AHMED EL-SHARKAWY
8. AHMED MORRA
9. AHMED NASEF
10. AHMED SHERIF
11. AKRAM AZZAM
12. ALAA GHEITA
13. ALAA MAHER
14. ALY MOFTAH
15. AMIR NASEF
16. AMR GAD
17. AMR LOTFY
18. AMR MOHSEN
19. AMR SAFWAT
20. ASHRAF FODA
21. AYMAN EL-SAMADONY
22. AYMAN SALAH
23. ESSAM EL-KOUSY
24. EZZ ELDIN KORASHI
25. GAMAL MOSTAFA
26. HANY RATEB
27. HASSAN SOLIMAN
28. HESHAM ABOU EISHA
29. IBRAHIM GALAL
30. IBRAHIM NAGEEB
31. KAIS A. ABULATA
32. KHALED ZOHDI
33. MAGDI EL-BARBARI
34. MAGED BARSOUM
35. MAHMOUD ABOU-ZEID

36. MAHMOUD EL-DAW
37. MAMOON ISMAIL
38. MEDHAT ASEM
39. MOHAMMAD ABDO AY EL-DIN
40. MOHAMMAD EL-BATANONY
41. MOHAMMAD KADRY
42. MOHAMMAD SABRY
43. MOHAMMAD SOBHI
44. MONGY ABD EL-KADER
45. MONTASSER EL-KOTBY
46. MOUSTAFA ABOUL NASR
47. MOUSTAFA EL-SHARKAWY
48. MOUSTAFA SOLIMAN
49. NABIL ABDEL MEGUID
50. NADR A. ABULATA
51. OMAR EL-KASHEF
52. RAAFAT AFIFI
53. RADY SAAD
54. SAFWAT ABDEL KADER
55. SAID RATEB
56. SALAH ABDEL GHANY
57. SALAH SHAHEEN
58. SALMA DAWARA
59. SAMIR A. GALAL
60. SAMIR SHALABY
61. SAYED ABDEL HALIM
62. SAYED MARIE
63. TAHER ISMAIL
64. TAREK ATTA
65. TAREK FAWZY
66. Wael GHAFEER

WOUND HEALING AND MANAGEMENT

Introduction

The ancient Egyptians were the first civilization to have trained physicians to treat physical ailments. Medical papyri, such as the Edwin Smith papyrus (circa 1600 BC) and the Ebers papyrus (circa 1534 BC), provided detailed information of management of disease, including wound management with the application of various potions and grease to assist healing.

Wound healing

Injury triggers an organized and complex cascade of cellular and biochemical events that result in a healed wound. The mechanisms and stages of wound healing are more or less the same in different tissues with minor variations.

Components of wound healing

1. **Wound contraction.** This process of contraction helps to diminish the size of the wound. It starts immediately after wounding and continues for the next 2-3 weeks. It is assumed to be due to special myofibroblasts, which contain the smooth muscle protein actin.
2. **Granulation tissue** formation, which is later replaced by fibrous tissue.
3. **Epithelialization.**

All three components share in healing of all wounds, but in a proportion and timing that differ according to the type of wound. Clean sutured wounds epithelialize in about 48 hours. Both wound contraction and granulation tissue/fibrosis occur to a little extent and at a later date. On the other hand if wound edges are wide apart, wound contraction and granulation tissue/fibrosis have larger contributions, while epithelialization covers the surface when the gap is filled to the surface with granulations.

Stages of wound healing

Wound healing response can be divided into three overlapping phases (Fig. 1.1).

1. Haemostasis and inflammation phase

- Injury of blood vessels in the wound leads to aggregation of platelets to the subendothelial collagen and activation of the coagulation cascade.

CHAPTER CONTENTS

- Wound healing
 - Components of wound healing
 - Stages of wound healing
 - Types of wound healing
 - Factors affecting wound healing
 - Complications of wound healing
- Types of wounds
 - Closed wounds
 - Open wounds
- Management of open wounds
- Suture materials
 - Absorbable sutures
 - Non-absorbable sutures
- Chronic wounds
- Wound dressing

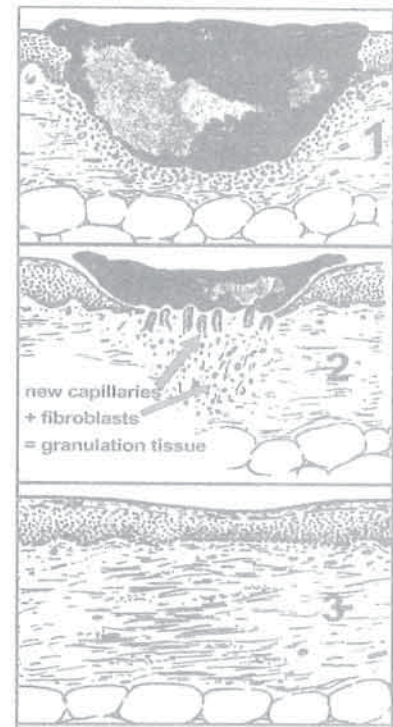


Fig. (1.1) Stages of wound healing

1. Inflammatory cells invade the wound area.
2. Granulation tissue formation and start of epithelialization.
3. Maturation of collagen and reorientation of fibres across the wound.

- From the platelets certain cytokines and growth factors are released, and these stimulate chemotaxis of leucocytes, macrophages and lymphocytes.
- Macrophages play an important role in phagocytosis and wound debridement. They release further growth factors, which lead to recruitment and activation of fibroblasts and endothelial cells.

This phase lasts for about 5 days, but it may be prolonged if there is wound infection.

2. **Proliferation phase** This phase is characterized by the proliferation of

- Fibroblasts which are derived from the surrounding tissues. They secrete collagen fibers.
- Endothelial cells which proliferate from the intact venules to form new capillary buds, which together with the fibroblasts form the granulation tissue.
- Epithelial cells which proliferate from the wound edges and injured epithelial islands within the wound. The basal cells of the epidermis start to undergo hypertrophy and mitosis and start migration to close the epithelial defect. In large open wounds the epidermis grows for few centimeters and then slows down and a raw area will persist. In any type of wound the young epithelium is thinner than normal epithelium and it possesses no sebaceous or sweat glands, nor hair follicles.



Fig. (1.2) A clean cut wound whose edges are coapted heals leaving a fine scar (primary intention).

3. **Maturation and remodeling phase**

- Deposition of collagen in the wound. Collagen III is deposited at first, but over the next weeks collagen III decreases while collagen I increases.
- Remodeling. With time the collagen fibers become thicker and they get arranged along the lines of stress leading to an increase in the tensile strength of the wound. The process of remodeling continues for about one year. It should be noted that a wound never attains its full original tensile strength.

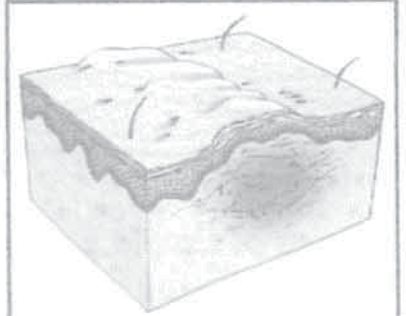


Fig. (1.3) A gaping wound heals by the formation of a large amount of granulation tissue that fills the gap. This is later replaced by fibrous tissue. The result is a wide ugly scar (secondary intention). A scar is devoid of sweat and sebaceous glands, and hair.

Types of wound healing

- **Healing by primary intention.** This occurs in clean wounds when they are immediately closed by sutures or clips. Healing occurs with minimal fibrosis leading to a nice neat scar (Fig. 1.2).
- **Healing by secondary intention.** This occurs when the wound edges are not approximated or when gaping occurs as a result of a haematoma or wound infection. Healing occurs by in-filling with granulation tissue and so there is more fibrous tissue. The resulting scar is ugly (Fig 1.3).

- **Healing by tertiary intention.** Wounds which are contaminated are left open for about 5 days. At the end of this period if there are no signs of infection, delayed primary sutures can be performed.

It is to be noted that many cells, growth factors and cytokines participate in the process of wound healing

Cells: Platelets, polymorphonuclear leucocytes, macrophages, lymphocytes, fibroblasts, endothelial cells and epithelial cells.

Growth factors and cytokines: Platelet derived growth factor, tumour necrosis factor-alpha, interleukin-I and fibroblast growth factor.

Factors affecting wound healing

General factors

1. **Age.** Wound healing is slow in elderly persons due to a reduced rate of protein turnover.
2. **Nutritional state.** Protein deficiency leads to diminished synthesis of collagen and ground substance. Vitamin C deficiency is responsible for lack of maturation of procollagen. Vitamin A deficiency leads to deficient epithelialization. Calcium, Zinc, Copper and manganese also affect wound healing.

- After one week the wound has only 3% of its final strength.
 - After three weeks the wound has 20% of its final strength.
 - After three months the wound has 80% of its original strength.

The tissues never regain their original tensile strength
3. **Debilitating diseases** as uraemia, jaundice, cirrhosis, diabetes and malignancy delay wound healing.
4. **Drug intake** Steroids, cancer chemotherapy and immunosuppressive drugs inhibit wound healing.

- Remember factors affecting wound healing when you study burst abdomen, incisional hernia and recurrent hernia.
 - Factors which favour wound infection include;
 - Foreign bodies
 - Dead tissues
 - Ischaemia
 - Suturing under tension

Local factors

1. **Vascularity.** A good blood supply, e.g., in the face and scalp, leads to nice healing while a poor blood supply (wounds below the knee) causes delayed healing.
2. **Irradiation** impairs wound contraction and granulation tissue formation as it causes ischaemia due to end arteritis obliterans.
3. **Immobilization.** Movement and shearing forces damage the blood supply of the granulation tissue. Immobilization, therefore, helps healing.
4. **Tension.** Any increased tension in the wound will lead to ischaemia and impaired healing. Sutures under tension, haematoma and infection increase tension inside the wound.
5. **Infection** delays healing. Fibroblasts compete with bacteria for oxygen and nutrition. Moreover, bacteria secrete collagenolytic enzymes, which destroy collagen fibers.
6. **Foreign bodies and necrotic tissue** impair wound healing.
7. **Adhesion** of the wound to a bony surface prevents wound contraction, e.g., wounds over the shin of tibia and chronic venous ulcers.

8. **Impaired venous drainage**, as in post-phlebitic limbs, impairs wound healing.

Complications of wound healing

1. **Wound failure (wound dehiscence).** General or local factors which are adverse to wound healing can lead to wound failure. Failure of an abdominal wound is called burst abdomen (Chapter 42).
2. **Stretching of the scar.**
3. **Hypertrophic scar.** The scar is raised above the surface but it remains within the confines of the wound. Within months it may regress. This problem is common in the shoulder and presternal area.
4. **Keloid formation.** There is over-activity of the healing process leading to excessive scar tissue which is raised above the surface, and extends beyond the confines of the original wound. It can follow burns, traumatic or surgical wounds, inflammation, ear-holing and vaccination.



Fig. (1.4) Keloid

Persons with dark skin are more prone to keloid formation (Fig. 1.4) and there is a familial predisposition. Certain areas as the ear lobules, shoulder and presternal areas are more liable to keloid formation.

Treatment of hypertrophic scars and keloids

- Continuous pressure by silicone gel sheets. Continuous pressure causes ischaemia of the small blood vessels leading to diminished activity of fibroblasts and diminished collagen synthesis.
 - Intralesional corticosteroids. Triamcinolone and a local anaesthetic are injected in the dermal region of the scar.
 - Surgical excision. Recurrence rate after simple excision may reach 80%. To minimize recurrence intramarginal excision of the scar is recommended together with intraoperative injection of steroids.
5. **Contracture.** This is pathologic shortening of scar tissue resulting in deformities if the scar overlies a joint (Fig. 1.5). Proper positioning of the joint during healing can minimize the deformity.
 6. **Surgical site infection.** (Chapter 7)



Fig. (1.5) Post-burn contracted scar of the neck

Types of wounds

Wounds may be either closed or open.

Closed wounds

1. **Contusions.** This is due to a blow with a blunt object leading to extravasation of blood through the injured capillaries. The injured area becomes painful and swollen. The contused area is at first bluish in colour due to the extravasated blood, later the colour turns to brownish or green. Elevation of the contused area is advisable to minimize oedema and a local anti-inflammatory ointment is applied.

2. **Haematoma.** If the amount of bleeding is excessive, a haematoma forms. At first it is cystic, but it will clot within hours. Later, the haematoma will liquefy. If a haematoma is small, it can be aspirated by a wide-bore needle, otherwise it is evacuated by a surgical incision under aseptic precautions.

Open wounds

1. **Abrasions.** An abrasion is scraping away of the superficial layers of the skin due to friction with a hard rough surface. The wound is very painful due to exposure of the sensitive nerve endings. Abrasions require cleaning with a bland antiseptic and a non-adherent dressing.
2. **Incised wounds.** These are produced by sharp cutting objects as a razor, a piece of glass or a knife. The wound is longer than deep, its edges are clean cut and there is no much tissue destruction. There is usually extensive haemorrhage; tendons and nerves are liable to be cut.
3. **Lacerated wounds.** These are caused by severe violence with blunt objects, e.g. road traffic accidents (RTA) or falling from a height. Such wounds are irregular in shape, and the tissues are severely traumatized and devascularized. They are usually contaminated and so the risk of infection is high. Inflammatory oedema will develop after a few hours and this will raise tension inside the wound (especially if the wound is closed) to high levels, leading to secondary ischaemia of the tissues. These wounds are commonly accompanied by a degloving injury where an area of the skin and subcutaneous fat are degloved from the underlying deep fascia. Devascularization of the skin will become slowly apparent in the following few days.
4. **Penetrating wounds.** These are caused by penetration of a pointed object as a knife. The wounds are more deep than long, so there is risk of injury to deep important structures. The external opening is small and drainage is poor, thus encouraging infection. These wounds are deceiving and an unwary surgeon may suture a stab wound in the abdominal wall and miss an injured viscus inside.
5. **Missile wounds.** These wounds are very serious as the bullet transmits its high kinetic energy to the tissues. The kinetic energy of the missile is determined mainly by its velocity, and secondly by its weight. Kinetic energy of a missile is calculated by the formula; **[Kinetic energy = $MV^2/2g$]** where M is the mass of the bullet, V is velocity, and g is the acceleration of gravity. From this equation, it is apparent that high-velocity missile injuries (rifles) are much more serious than low-velocity ones (pistols). This damage is the result of the following factors
 - a. Direct damage by the missile in its track.
 - b. Shock waves that precede the missile in its journey through the body. Sometimes, damage occurs in areas far away from the missile tract.
 - c. Temporary cavitation effect.
 - d. If the missile strikes a bone, the fragments of shattered bone serve as secondary missiles producing more damage. In high velocity injuries there is extensive tissue damage and injury to the major blood vessels and nerves situated some distance from the tract of the missile.
6. **Bites.** These may be animal or human bites. They are lacerated and contaminated wounds with a high incidence of infection.

Remember

1. In penetrating wounds, always suspect injury to deep structures.
2. In missile injuries, structures far away from the site of the missile tract may be injured.

Management of open wounds

1. Follow the priorities of management of multiple-injury patients (Chapter 2).
2. Inquire about the timing and the cause of injury. One can form an idea about the possible injured structures from the cause and site of injury; e.g. a stab wound in the femoral triangle may injure the femoral vessels. In missile injuries, structures far from the tract of the missile may be injured.
3. Prophylaxis against tetanus (Chapter 7).
4. Prophylactic antibiotics are prescribed. In deep or lacerated wounds, antibiotics against anaerobic infections should be added.
5. If there is bleeding from the wound, the best way to stop it is by direct local compression by applying a sterile dressing and tight bandage. Do not apply a tourniquet except as a temporary measure before taking the patient to the theater.
6. If a fracture is suspected, arrange for a plain X-ray and splint the fracture.
7. Feel the distal pulses, if not palpable reassess after treatment of shock. Urgent duplex is mandatory if the pulses are not palpable.

Management in theatre

1. Thorough **cleaning** of the wound by saline irrigation is performed. Meticulous removal of foreign bodies is very important, as they constitute a very fertile medium for microorganisms. Sterilize the wound by an antiseptic, e.g. povidone iodine (Betadine). Inspect the wound and deal with every injured structure.
2. If a major **artery or vein** is injured, it should be repaired according to the usual principles. A small bleeding vessel can be ligated.
3. Injured **nerves or tendons** can be repaired in clean incised wound but in contaminated wounds it is better to defer the repair.
4. If there is a clean incised injury of a **muscle**, it can be repaired by mattress sutures. Ischaemic or necrotic muscles should be completely excised. Necrotic muscle is dark red or gray in colour, it does not contract if pinched, and it does not bleed if incised.
5. In contaminated wounds or where there is extensive tissue destruction, the deep fascia should be left open.

6. Skin

- **Clean and early** (<6 hours) primary closure.
- **Contaminated or late** delayed primary closure.
- **Lacerations** debridement to leave healthy vascularized tissues.



Fig. (1.6) Degloved skin injury of the foot

- In degloved **skin injuries** (Fig. 1.6) where the viability of the skin is doubtful, defer the decision to a second look after 3-4 days. In these circumstances the deep fascia and skin should be left open. If there is a skin defect, the patient will be reassessed later when the tissues are healthy, then skin graft or flap may be performed.

- If there is an associated **fracture** and there is any possibility of infection, it is advisable to stabilize the fracture by some form of external fixation as it is inadvisable to resort to internal fixation in these circumstances.
- All **missile** injuries require exploration.

N.B. In contaminated wounds 1. Do not do nerve repair. 2. Do not do tendon repair. 3. Do not close the deep fascia. 4. Do not close the skin.

Suture materials

Sutures are used to approximate wound surfaces until healing occurs. Suture materials are classified according to the following

- Their origin; natural or synthetic.
- Their absorption; absorbable or non-absorbable.
- Their mode of synthesis (**Fig. 1.7**); monofilamentous or multifilamentous (braided).



Fig. (1.7) Monofilament and braided sutures

In general, natural suture materials initiate more tissue reaction and can augment and maintain infection more than synthetic materials. Multifilamentous sutures induce more tissue reaction than the monofilamentous. Introduction of a multifilament suture in the presence of infection can pick up 10 times more organisms than the equivalent monofilament. The capillary spaces between the filaments allow the organisms to settle.

Absorbable sutures

1. **Chromic catgut** is a modified form that is prepared from plain catgut by chroming. It is absorbed within 3 weeks and is less irritant than plain catgut. It is used for anastomosis in the gastrointestinal and urinary tract. Chromic catgut loses 50-80% of its strength in two weeks.
2. **Polyglycolic acid (Dexon)**
 - a. Multifilamentous.
 - b. Has good tensile strength.
 - c. Absorption occurs between 2-3 months.
3. **Polygalactin (Vicryl)**
 - a. Multifilamentous.
 - b. Absorption occurs after 4 months.
 - c. It can be used for gastrointestinal anastomoses.
4. **Polydioxanone (P.D.S.).** Monofilamentous. It loses 50% of its tensile strength in 6 weeks.

Non-absorbable sutures

1. **Silk**
 - This is a protein material. It, therefore, stimulates a marked tissue reaction.
 - Multifilamentous.
 - Easy handling.
 - If infection occurs silk sutures should be removed to allow infection to subside.
 - Silk loses 40% of its strength in 6 weeks.
2. **Polyamide (Nylon)**
 - Mono or multifilamentous.
 - Good tensile strength.
 - The knot is not very secure.

3. Polypropylene (Prolene)

- Monofilamentous.
- Good tensile strength.
- Very inert and even if infection occurs, there is no need to remove the sutures.
- Difficult knotting.
- Ideal suture material for closure of abdominal wounds and for hernia repair.

4. Stainless steel

- Mono or multifilamentous.
- Very high tensile strength.
- Very inert.
- Difficult handling.
- Used for closure of sternotomy in open heart surgery.

Other methods of skin closure

1. Clips
2. Staplers (**Fig. 1.8**)
3. Adhesive tapes can be applied alone or to supplement sutures. They cause no inflammatory reaction as no suture material transverses the skin.

Chronic Wounds

Chronic wounds fail to undergo the usual sequence of acute healing within a reasonable time, usually within three months. The forementioned factors affecting the healing process act singly or in combination to impair the normal healing e.g. local ischaemia, venous congestion, diabetes, steroids or the presence of necrotic tissue. Most of chronic wounds are arrested at the inflammatory and proliferative phases with overgrowth of granulation tissue with minimal wound contraction and collagen formation.

In the management of chronic wounds, the general factors affecting the healing process should be managed hand-in-hand with local wound management. Most chronic wounds share local hypoxia due to ischaemia, trauma, vasculitis among other factors.

Of the heterogeneous group of chronic wounds, three categories are mentioned:

- **Leg ulcers** An ulcer is defined as a break in the continuity of epithelium or mucous membranes. The commonest leg ulcers are the **venous** ulcers. Other types of leg ulcers are ischaemic ulcers, neuropathic ulcers, neoplastic ulcers due to squamous cell carcinoma or basal cell carcinoma (rare).
- **Diabetic foot ulcers** are described in chapter (13).
- **Pressure sores** (bed sores or decubitus ulcers). Pressure sores usually develop in bedridden patients due to high level of pressure applied to the skin.
 - Risk factors Elderly patients, poor nutrition, anaemia, denervation, paraplegia, ICU patients, immobilization, and skin soiling by stools or urine.
 - Common sites Ischium, greater trochanter, sacrum, heel and malleoli.
 - Stages (4)
 1. Erythema
 2. Partial thickness skin loss.
 3. Full-thickness skin loss but short of the fascia.
 4. Same as 3 with further tissue destruction; muscles, bone and joints.
 - Prevention and treatment
 - i. Correction of general factors as malnutrition and skillful nursing care as turning in bed.

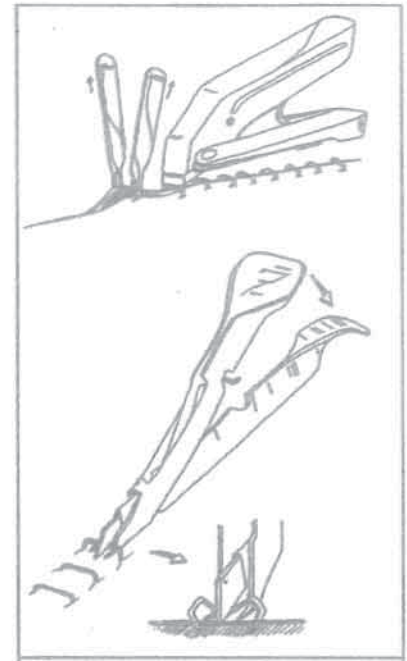


Fig. (1.8) Applying skin staples and removing them

- ii. Assure a dry skin.
- iii. Protect pressure areas by cushions or foam.
- iv. The use of air mattresses.
- v. Regular surgical debridement and the use of vacuum assisted closure (VAC).
- vi. Rotation flaps, myocutaneous or fascio-cutaneous flaps.

Wound Dressings

The main purpose of wound dressings is to provide the ideal environment for wound healing and prevent bacterial overgrowth.

Some of the common forms of wound dressings include

- Occlusive dressings as conventional gauze with adhesive plaster afford wound protection and prevent undue dryness of the wound by preventing evaporation thus a reasonable level of hydration is maintained. Local hypoxia of the wound stimulates capillary proliferation and favours autolysis of the sloughs. Occlusive dressings are not suitable for infected and highly exudative wounds.
- Semioclusive dressings are permeable to water vapour and gases and impermeable to bacteria. Some are available as hydrocolloid sheets affording a moist environment beneath the dressing for optimum wound healing.
- Alginates are derived from seaweeds. On contact with the wound exudates, a fibrous gel forms and creates a moist environment.
- Osmotic agents as Debrisan, urea crystals or honey absorb the exudates.
- Two factors have been shown to enhance the whole healing reactions
 - o Vacuum assisted closure (VAC) affords a negative pressure environment which enhances the formation of granulation tissue and stimulates wound contraction. It has shown good results in bed sores and diabetic ulcers.
 - o Increased local hydration of the wound affords a moist environment (moist healing).

MAJOR TRAUMA AND THE MULTIPLE-INJURY PATIENT

Introduction

Trauma is a common cause of mortality both in civilian life and during war time. It is the leading cause of death for individuals of age 1-44 years, and ranks third in causing mortality in all ages. In addition, it is a major cause of morbidity. For every trauma death, two people suffer permanent disabilities.

Major trauma commonly causes multiple injuries in different parts of the body.

Mechanism of injury

There are two major types of injuries

1. Penetrating injuries

- Low velocity injuries. These are caused by
 - Knives, spikes of glass, and other sharp objects.
 - Bullets that are fired by pistols travel at a slow velocity and are, therefore, included in this group. The injury is usually focused over a small area.
- High velocity injuries. The common example is firearm injuries that are caused by rifles. Here the energy may be dissipated over a wide area. Shock waves spread out from the main missile tract and affect areas far from the primary missile tract. The higher the velocity of the missile, the more is the damage it causes (chapter 1).

2. Blunt injuries

- Direct blows
- Fall from a height
- Road traffic accidents (RTA)

When a pedestrian is struck by a moving vehicle, there is often an acceleration injury in addition to the direct trauma at the site of impact. A person inside a moving car acquires the same velocity of the car. If the car stops suddenly, the person will continue moving forwards and if he is not wearing the seat belt, the head will strike against the car. The force of impact of the body against the seat belt may itself cause fracture of the clavicle, damage to the

CHAPTER CONTENTS

- Introduction
- Mechanism of injury
- Causes of trauma mortality
- Organized trauma care
- Primary survey/resuscitation
- Secondary survey
- Definitive treatment of individual injuries

Trauma care principles

- There is a need for rapid evaluation of the trauma patient. Time wasted costs lives.
- The absence of a definitive diagnosis should never impede the application of lifesaving measures.
- Management in the first 'Golden Hour' is crucial to both the short and long term survival of the patient. It is also critical in determining the morbidity that the patient will endure.
- There is a need to establish management priorities. The things which will kill the patient first are always the things which should be checked and treated first. Things which will kill the patient later are managed later. Thus, airway problems are managed and treated before breathing problems, which in turn are treated before circulatory problems.



Fig. (2.1): The seat belt mark. Suspect seat belt injuries

small intestine, the mesentery, stomach or duodenum, the so-called seat belt injuries. The skin mark of the seat belt should raise suspicion of such injuries (Fig. 2.1). It should be noted, however, that the protective benefits of a seat belt far exceed its possible injurious effect.

With blunt trauma there is a tendency for certain patterns of associated injuries, e.g., a combination of head and cervical injuries, sternal and dorsal spine injuries, fracture of the lower ribs and rupture of the liver or spleen, and pelvic fracture with bladder or urethral injuries.

Causes of trauma mortality

Deaths following trauma can be classified into 3 groups according to the timing after the accident.

Immediate deaths

These follow fatal injuries and occur within few minutes after the accident so that little can be done for the victims. Examples of these injuries include major trauma to the brain or upper spinal cord, injuries of the heart or major blood vessels or rupture of the major airway.

Early deaths

These occur within few hours after the accident and so, with proper and rapid management, the patients have a chance of survival. These cases include intracranial haemorrhage, massive intra-abdominal or intrathoracic haemorrhage, or major fractures.

Late deaths

These occur some weeks after the injury, generally due to sepsis or multiple organ failure.

Organized systems for trauma care are focused on saving a trauma victim from early mortality, while critical care is designed to prevent late trauma mortality.

Organized trauma care

- Victims of major trauma are best treated by a well-organized and trained trauma team.
- Accident and emergency departments should have an equipped resuscitation area set aside to receive major trauma victims.
- In mass casualty accidents, e.g., train accidents or earthquakes, the concept of triage is important. Triage means sorting of patients, i.e., their ranking according to both their clinical need and the available resources to provide treatment. It may take two forms
 - If the number of casualties does not exceed the facilities all critically injured are treated.
 - If the number of casualties exceeds facilities, then the critically injured most likely to survive are treated first.
- The American College of Surgeons developed the Advanced Trauma Life Support (ATLS) which is an internationally accepted protocol for the management of major trauma victims. ATLS protocol has three elements
 - Primary survey resuscitation.
 - Secondary survey.
 - Definitive treatment of individual injuries. This will be discussed in the corresponding chapters.

ATLS

1. Primary survey/resuscitation.
2. Secondary survey.
3. Definitive treatment.

Airway obstruction may kill the trauma victim within minutes. Its relief should receive top priority.

- The primary survey and resuscitation should start at the site of accident by trained ambulance personnel. It continues as the trauma victim reaches the hospital.

Primary survey/resuscitation

Objective

The objective is to identify and treat any immediately life-threatening condition.

Steps

In sequence the five steps of the primary survey are A, B, C, D, E.

- Airway (and cervical spine control).
- Breathing.
- Circulation with haemorrhage control.
- Disability (brief neurological assessment).
- Exposure and Environment.

Life-threatening problems discovered during the primary survey are always dealt with before proceeding to the secondary survey.

A. Airway (and cervical spine control)

The patient's airway is evaluated and protected if necessary, while concomitantly controlling movement of the cervical spine. In general, if the patient is capable of unstrained speech, his airway is patent. All patients receive supplemental oxygen by mask upon arrival.

Clear the airway

1. Vomit, blood or foreign material should be removed manually (finger sweep) or with a rigid sucker.
2. This is followed by chin lift or jaw thrust (Fig. 2.2).

Protect the airway

- An oropharyngeal or nasopharyngeal airway tube prevents the tongue from falling back and occluding the airway in an unconscious person.
- Tracheal intubation is indicated with
 - Apnoea
 - Risk of aspiration
 - Impending or actual airway compromise (inhalation injuries, maxillofacial trauma)
 - Closed head injuries (allow hyperventilation to decrease intracranial pressure [ICP])
 - Orotracheal intubation allows the use of a large tube.
 - Nasotracheal intubation is safer if the cervical spine appears fractured.
- Cricothyroidotomy (Fig. 2.3). This is done either by making a cut and inserting a tube, or by the percutaneous insertion of a wide-bore needle. This procedure is not suitable for children.

N.B. Tracheostomy is rarely needed in the emergency room management.

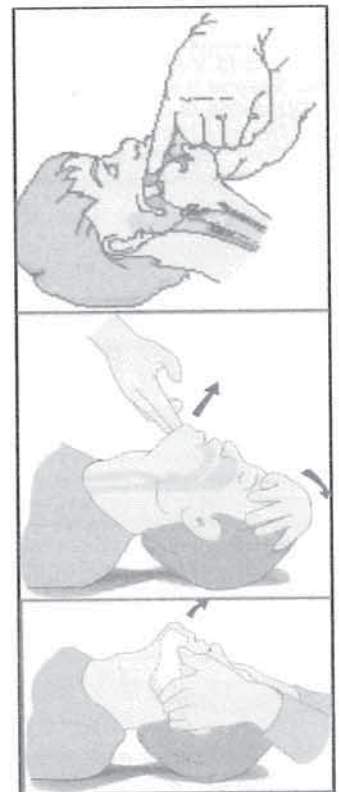


Fig. (2.2) Clear the airway

- Finger sweep
- Chin lift



Fig. (2.3) Cricothyroidotomy

Cervical spine control

The cervical spine should be considered unstable (pending radiological diagnosis) in the following situations

- Clinical examination reveals bony abnormalities or cervical tenderness.
- Multisystem trauma, a blunt injury above the clavicle, or an altered level of consciousness from trauma or from drug/alcohol intake.
- Maxillofacial trauma.

Cervical spine immobilization is done using a backboard and a rigid collar. If a collar is not available, manual in-line immobilization is necessary (Fig. 2.4).

Radiological evaluation is done later after stabilization of vital systems. At least three views of the cervical spine (lateral, AP, and odontoid) are done.

B. Breathing

Assessment

1. Inspection for air movement, respiratory rate, cyanosis, tracheal shift, jugular venous distention, open chest wounds, asymmetric chest expansion and use of accessory muscles of respiration.
2. Palpation for subcutaneous emphysema and flail segments.
3. Auscultation for upper airway sounds (stridor, wheezing, or gurgling) and for lower airway sounds present over lung fields.
4. Percussion for hyperresonance or dullness over either lung field.

The immediately life threatening thoracic conditions and their treatment are

1. Tension pneumothorax. Needle decompression followed later by intercostal chest tube (Fig. 2.5).
2. Cardiac tamponade. Needle pericardiocentesis (Fig. 2.6) followed later by operative pericardiotomy and control of source of bleeding.
3. Flail chest caused by fractures of adjacent ribs. This is commonly associated with lung contusion. Treatment is by intubation and positive pressure ventilation.
4. Massive haemothorax. Initial treatment is by chest tube insertion to allow lung expansion. Later thoracotomy, may be needed if bleeding continues.
5. Open pneumothorax. Initial treatment is by an occlusive dressing fixed at 3 sides only followed by insertion of a chest tube.

C. Circulation

Shock

1. Haemorrhagic. Commonest. Tension pneumothorax reduces venous return and worsens this shock.
2. Cardogenic. Tamponade and myocardial trauma.
3. Neurogenic. Spinal cord injury.

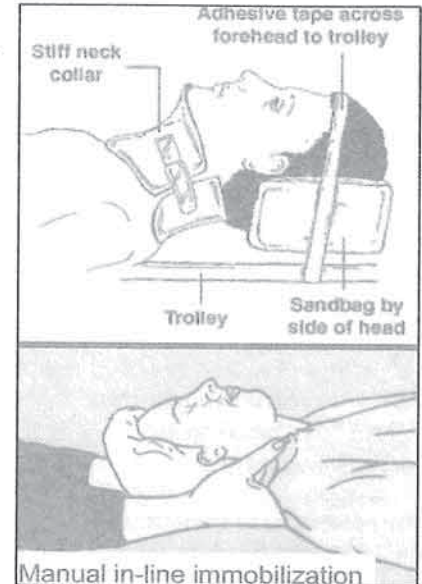


Fig. (2.4) Cervical spine immobilization

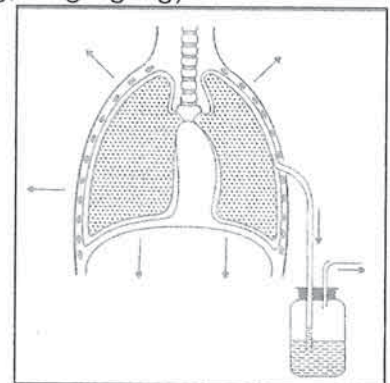


Fig. (2.5) An intercostal chest tube is used to drain air or fluid from the pleural cavity. The tube is connected to an under-water seal

Action

1. Bleeding is controlled with direct pressure if possible. Sites of hidden haemorrhage are intra-abdominal, intra-thoracic, and fractures of pelvis and femur.
2. Two large-calibre (16 gauge) peripheral IV lines are inserted. A central line may also be added.
3. Blood samples are sent for typing, cross-matching, haemoglobin, haematocrit and blood chemistry.
4. Ringer's lactate solution is infused as a start. Volume of crystalloid needed = 3 X estimated blood loss.
5. When cross-matched blood is available it is to be infused immediately. IV fluids and blood are given at a rate that ensures an optimum urine output of 0.5-1 ml/kg/hour for adults.

**D. Disability** (brief neurological assessment)

Common causes of neurological deficits related to trauma are

- Head injury.
- Hypoxia.
- Shock.
- Alcohol or drugs abuse.

Fig (2 6) Pericardiocentesis for aspiration of blood from the pericardium. The needle is inserted to the left of xiphoid and is directed to the tip of left scapula.

Management priorities in major trauma cases are Airway, Breathing, Circulation, Disability, and Exposure

AVPU evaluation

Based on patient's best response.

- A. Alert and interactive
- V. Vocal stimuli elicit a response
- P. Painful stimuli are necessary to evoke a response
- U. Unresponsive

This provides only brief neurological information. A more detailed assessment using the Glasgow Coma Scale (GCS) is performed during the secondary survey.

E. Exposure and environment

Clothes All clothes of the trauma victim are removed using sharp large scissors.

Warmth Keeping the emergency room warm and using blankets to prevent hypothermia,

Insert

- Urethral catheter (Foley) to monitor urine output. This is contraindicated if there is blood at the urethral meatus as it indicates urethral injury. Trial of a catheter insertion in this case is usually unsuccessful and may even compound the injury.
- Nasogastric tube (Ryle's) decompresses the stomach and prevents vomiting and aspiration.



Fig. (2.7) Cervical spine X-ray shows fracture dislocation between C5 and C6.



Fig. (2.8) Chest X-ray of a major trauma victim shows a widened mediastinum which denotes aortic rupture

Radiological assessment

- For blunt trauma cervical spine (Fig. 2.7), AP chest (Fig. 2.8) and AP pelvis (Fig. 2.9) X-rays are mandatory.
- For penetrating trauma, AP chest and X-ray of trauma site, if applicable.
- After resuscitation other X-rays or CT scans are performed as indicated.

History

The patient's history should be obtained while completing the primary survey. If the patient is unconscious, the rescue team, accident witnesses, and family members should be relied upon to get the following information that have the acronym AMPLE.

- A. Allergies
- M. Medications
- P. Past medical history
- L. Last meal (time)
- E. Events of injury.

Secondary survey

The secondary survey is to be done once resuscitation efforts are under way and preliminary X-rays have been evaluated.

Objectives

1. Examination of the patient from head to toe and front to back.
 2. Taking a complete medical history.
 3. Integration of all clinical, laboratory, and radiological information.
 4. Formulation of a management plan.
- It includes examination of
1. Head. Haematoma, fractures and pupils.
 2. Face
 3. Spine. To allow examination of the back the patient's body is turned in one piece (log rolling) by four persons. This is to avoid injury of the spinal cord if there is an unstable spine fracture (Fig. 2.10).
 4. Neck
 5. Chest
 6. Abdomen For indications of abdominal exploration see chapter 40. CT (Fig 2.11) or Diagnostic peritoneal lavage (DPL) are indicated in blunt abdominal trauma, in an adult, that is associated with
 - Suspicion of organ injury with equivocal signs.
 - Unreliable abdominal examination because patient is unconscious, e.g., head trauma, or drug or alcohol intoxication.
 - Unexplained hypotension that may be caused by blood loss. The technique, interpretation and limitations of DPL are discussed in chapter 40.
 7. Perineum, including rectal examination in all patients and vaginal examination in females.
 8. Limbs for fractures and for soft tissue injuries including vessels, nerves, and tendons,

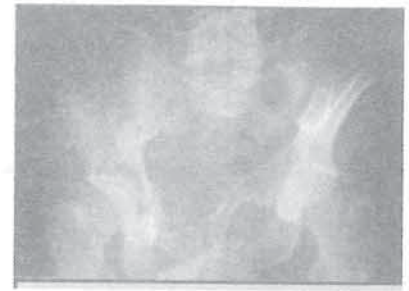


Fig. (2.9) X-ray of the pelvis shows an unstable fracture of the pelvis, which is likely to be accompanied by bladder or posterior urethral injury

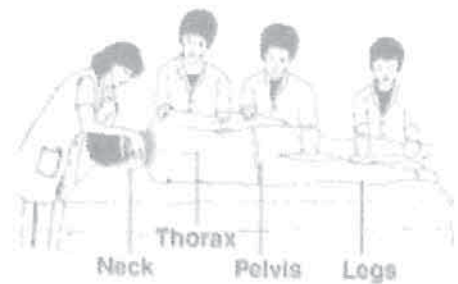


Fig. (2.10) Log rolling of a trauma victim

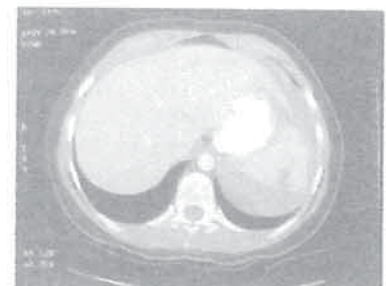


Fig. (2.11) CT scan of abdomen shows a tear in the spleen. The lower picture shows the splenic injury at abdominal exploration.

9. Nervous system

- Pupils for size, equality, and reaction to light.
- Glasgow Coma Scale (see chapter 19).
- Cranial nerves.
- Sensation and motor activity in limbs.
- CT scan of head if there is suspicion of intra-cranial injuries (Fig. 2.12).

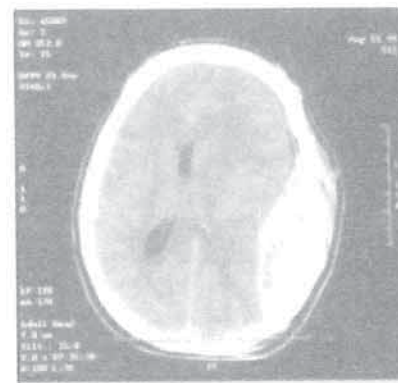


Fig. (2.12) CT scan of head shows an extradural haematoma

Definitive treatment of individual injuries

This will depend upon the type of injury and will be discussed in the relevant chapters. During this phase the following should be noted

- Some cases may require transfer either to another hospital with specialized facilities, or to another department in the same hospital.
- The level of care should not be allowed to drop during the transfer.
- The patient requires repeated evaluation as some injuries may present late after the accident, e.g., some injuries of the spleen, retroperitoneal duodenal injuries, and subdural haematoma.

Points to remember

The fives of major trauma

At the end of the major survey, detect or exclude the following five serious problems

- Airway obstruction.
- Pneumothorax, open or tension
- Massive haemothorax
- Flail chest
- Cardiac tamponade

When you face a patient with severe bleeding, remember the five areas of potentially severe bleeding

- Chest
- Abdomen
- Pelvis
- Long bone fractures
- External bleeding

The five causes of upper airway obstruction

- Tongue
- Blood
- Loose teeth or denture
- Vomitus
- Soft tissue edema

Five main features of respiratory distress

- Tachypnea and/or dyspnea
- Use of accessory muscles of respiration
- Difficult speaking
- Low oxygen saturation at bedside oximetry e.g. 80%
- Agitation or confusion

- In analyzing the causes of death in major trauma, 3 interrelated factors (hypothermia, coagulopathy and metabolic acidosis) make a vicious circle leading to a potentially fatal outcome Hypothermia; leads to slowing of the clotting factors and coagulopathy. Warming of the patient by a favourable external temperature, blankets or the use of warm i.v. fluids is essential. Coagulopathy in turn leads to diminished tissue perfusion, exaggeration of the shock state and metabolic acidosis due to anaerobic metabolism.

FLUID, ELECTROLYTE BALANCE AND ACID-BASE REGULATION

Claude Bernard was the first to recognize that to function effectively, the body needs a stable 'milieu interieur'. That 'milieu' is mainly water in which certain inorganic salts (electrolytes) are dissolved, that keep a constant body osmolality. A very delicate acid-base regulating mechanism is also crucial for life, since most enzymatic processes operate only within a narrow pH range.

Water, electrolyte, and pH imbalances rarely occur in pure form or in isolation; clinical problems are mixtures. In this chapter, for purposes of understanding and planning therapy, it was found useful to categorize clinical abnormalities.

Physiological considerations

Body water

- The total body water varies from 45-75% of the body weight, depending on the body content of fat. An adult male contains 60% water; a female having more fat contains 55% water, while a newborn infant has 75% water.
- The total body water is divided into intracellular (2/3) and extracellular (1/3) compartments. The extracellular fluid (ECF) is further subdivided into the interstitial compartment and the intravascular compartment which is the plasma (Fig. 3.1).
- Water freely distributes through all body compartments to bring the osmolarity of all compartments into equilibrium.
- The sources of water in the body are either exogenous or endogenous. Exogenous water intake is provided either by drinking or ingestion of solid food. The input varies widely, but averages 2-3 litres per day, of which slightly less than half is contained in solid food. Endogenous water is released during the oxidation of carbohydrates to water and carbon dioxide, and is known as metabolic water which amounts to 350 ml water pre day.
- This total water input is normally balanced with the water output from the body. Water is lost from the body by four routes (lungs, skin, faeces and urine).
 1. Lungs. About 400 ml of water is lost in expired air each 24 hours and is greater with increased respiratory rate.
 2. Skin. Water is lost through the skin by two mechanisms; an obligatory insensible perspiration containing virtually no sodium and an active sweating rich in sodium

CHAPTER CONTENTS

- Physiological considerations
 - Body water
 - Electrolyte metabolism
 - Acid-base regulation
- Water imbalance
 - Water depletion
 - Water excess
- Electrolyte imbalance
 - Hyponatraemia
 - Hypernatraemia
 - Hypokalaemia
 - Hyperkalaemia
 - Calcium imbalance
- Acid-base imbalance
 - Metabolic acidosis
 - Metabolic alkalosis
 - Respiratory acidosis
 - Respiratory alkalosis
- Practical applications
 - Diagnosis of imbalances
 - Postoperative fluid and electrolyte therapy

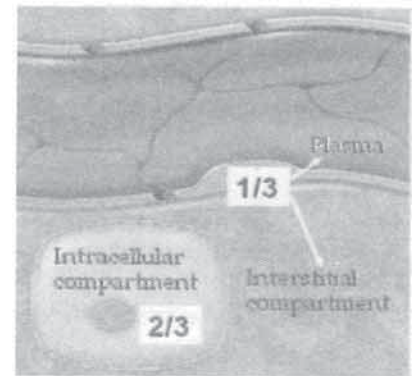


Fig. (3.1) Distribution of body water.

- Two thirds are intracellular.
 - One third is extracellular.
- Water = 60% of body wt.
 Intracellular = 40% of body wt.
 Extracellular = 20% of body wt.
- | | |
|--------------|-----|
| Interstitial | 15% |
| Plasma | 5% |

depending mainly on climatic conditions. In a temperate climate, the average insensible obligatory water loss is between 600-1000 ml per day.

3. Faeces. Between 60-150 ml of water are lost by this route daily and is increased with diarrhea.
4. Urine. The output of urine is under control of multiple influences, such as blood volume, hormonal and nervous factors, the most important of which is the antidiuretic hormone (ADH). The amount of water excreted in urine varies after the previous three routes have been met. The normal urinary output is approximately 1500 ml/day. A minimum urinary output of about 400 ml/day is required to excrete the end products of protein metabolism.

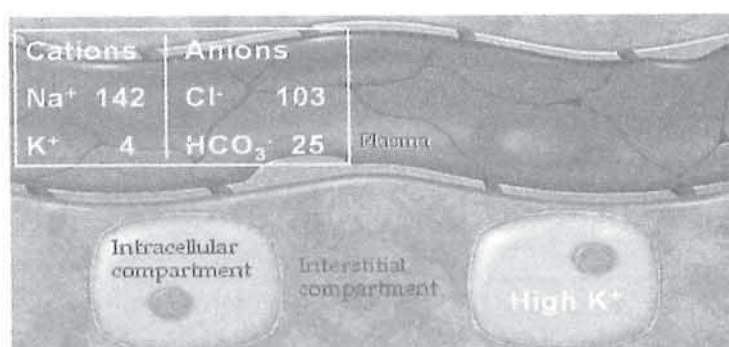


Fig. (3.2) Extracellular electrolyte levels

Significance of serum electrolyte estimation

- Na⁺ tells you the state of water balance, e.g., a high serum sodium indicates pure water loss.
- K⁺ tells you the patient's serum potassium status. This must be kept as close to normal as possible.
- HCO₃⁻ tells you if the patient has metabolic acidosis or alkalosis.

Electrolyte metabolism

Sodium is the main extracellular cation and potassium is the main intracellular cation. The normal serum electrolyte levels (Fig. 3.2) are roughly

▪ Cations	Na ⁺	142 mmol/L	K ⁺	4 mmol/L
▪ Anions	Cl ⁻	103 mmol/L	HCO ₃ ⁻	25 mmol/L

Under normal conditions, the number of anions must equal the number of cations to keep the electrochemical neutrality of the ECF. The main cations are sodium and potassium, while the main anions are chloride, bicarbonate, phosphates, sulphates, proteins and organic acids. Practically HCO₃⁻ and Cl⁻ are the anions that are measured in the laboratory. Thus, when we add the normal values for these, they are less than those of the cations. The difference is known as the anion gap, and represents the other anions that are not usually measured.

The interstitial fluid content is similar to plasma but with very low protein and a slightly higher chlorides.

Sodium

- Sodium is the main extracellular cation. It plays a central role in maintaining blood volume.
- There is normally a balance between sodium intake in the diet and its output mainly in urine, some in faeces and a negligible amount in insensible perspiration. However, in hot climate, profuse sweating results in a considerable loss of sodium.
- The average daily intake of sodium is 1 mmol/kg, which is about 5 gm/day, equivalent to 500 ml of isotonic 0.9% saline solution.
- Sodium balance is largely controlled by regulating its output which is governed by the variation in the avidity with which the renal tubules reabsorb sodium from the glomerular filtrate and the amount of sodium excreted by the sweat glands. This is under the control of the adrenal corticoids; the most powerful conservator of sodium being aldosterone.

- Obligatory reduction in sodium excretion follows surgery or trauma for a period of about 48 hours due to increased adrenocortical activity. It is inadvisable to administer large quantities of isotonic saline (0.9%) solution immediately after surgery for about two days.

Potassium

- Potassium is the major intracellular cation.
- Abnormalities of potassium concentration are of concern because of the risk of cardiac arrhythmias.
- The normal daily potassium intake is about 1 mmol/kg, mainly in potassium-rich food such as fruit, milk and honey.
- Potassium excretion is mainly in urine and almost equals the intake. A very small quantity is lost in formed faeces and still a smaller quantity in sweat.
- Augmented potassium excretion follows surgery and trauma. There is a period of increased excretion of potassium by the kidneys varying directly with the degree of tissue damage. This loss is greater during the first 24 hours and lasts for about 3-4 days. So great are the reserves of potassium that, unless the patient is severely depleted at the time of operation, hypokalaemia may not reveal itself for 48 hours.

Calcium

- The majority of body calcium is found in bones in form of phosphate and carbonate. The remainder is present as an extracellular cation which exists in almost equal two forms
 1. Ionized free fraction, which is responsible for the biologic effects of calcium, such as the neuromuscular stability, blood coagulation and cellular enzyme processes.
 2. Non-ionized protein-bound, chiefly albumin.
- Determination of the plasma protein level is essential for proper analysis of the serum calcium level. A low albumin level gives a false low total serum calcium concentration and vice versa.
- The ratio of ionized to non-ionized calcium is related to the pH; acidosis causes an increase in the ionized fraction, whereas alkalosis causes its decrease; respiratory alkalosis due to hyperventilation results in tetany with an apparently normal total serum calcium level.
- The normal serum level is between 8.5 and 10.5 mg/dl (4.25-5.25 mEq/L or 2.2-2.5 mmol/L).
- The serum calcium level is likely to be modified by vitamin D, parathormone, calcitonin and the state of renal and small bowel function.

Acid-base regulation

The normal ECF pH is 7.4 ± 0.04 , and most enzymatic processes operate only within a narrow pH range (7.3-7.5). Normal metabolism produces approximately 15000 mEq H^+ /day. Mechanisms for the regulation of body pH can be divided into three categories

- **Body buffer systems (pH shock absorbers):** The most important of these systems is the bicarbonate-carbonic acid ratio, which is normally 20:1. Alteration in this ratio changes the pH regardless of the absolute values of the bicarbonate and carbonic acid. A decrease in the ratio leads to increased acidity and vice versa. The bicarbonate level can be changed by metabolic factors, while carbonic acid level is subjected to alteration by respiratory factors. Alteration of one is followed automatically by a compensatory change in the other in an attempt to keep this ratio and pH constant.

- **Renal regulation (works in hours and days)** These function to maintain body pH by:
 - - Reabsorbing filtered bicarbonate.
 - Excreting 50-100 mmol of H^+ /day.
 - Renal failure is associated with a state of chronic acidosis.
- **Respiratory regulation (works within minutes):** This occurs by elimination of CO_2 , which is the source of carbonic acid. Since CO_2 diffuses approximately 20 times more readily than O_2 , severe respiratory impairment must occur before CO_2 retention occurs.

Water imbalance

Water depletion (pure dehydration)

Pure water depletion is rare in surgical practice.

Aetiology

1. Lack of water intake. This may be due to lack of availability, difficulty to swallow or inability to swallow in comatosed patients.
2. Diabetes insipidus.
3. Increased output in fever and osmotic diuresis.

Consequences

- A water deficit results in a decrease in volume of all the body fluid compartments.
- Since solute content does not change, hyperosmolality results.
- Osmoreceptors are stimulated, and secretion of ADH is increased. More water is reabsorbed from the distal renal tubules.

Clinical features

1. The main symptoms are intense thirst and weakness.
2. Reduced tissue turgor.
3. Oliguria with high specific gravity.

Treatment

1. An initial estimate of the magnitude of water deficit can be made from clinical data and by assuming that each 3 mmol elevation of serum sodium concentration above normal range represents a deficit of approximately one litre of body water.
2. Treatment of a water deficit requires provision of additional sodium-free water, e.g., IV 5% glucose.

Water excess (water intoxication)

Aetiology

Water excess often is iatrogenic, resulting from administration of electrolyte-free water.

1. The commonest cause on surgical wards is over-infusion of intravenous 5% glucose solution to postoperative patients.
2. Colorectal washouts with plain water instead of saline before colonic surgery.
3. A major component of the transurethral resection of the prostate (TURP) syndrome is the water intoxication caused by excessive uptake of water from irrigation fluid. In current practice water is replaced by glycine to overcome this problem.

Consequences

- Water excess leads to an increase in the volume of all fluid compartments.

- Since body solute content is not altered, a state of hypo-osmolality results.
- Hypothalamic osmoreceptors are inhibited, and pituitary secretion of ADH is decreased resulting in increased renal water excretion.

Clinical features

- Moderate water excess is often well tolerated and asymptomatic. The only findings are
 - Increased urine volume.
 - An increase in body weight. Pitting edema usually does not develop.
 - Decreased serum sodium concentration and falling haematocrit value.
- Marked water excess causes (serum sodium concentration is below 120 mEq/litre)
 - Swelling of brain cells that leads to drowsiness, weakness, and ultimately convulsions and coma (Water intoxication).
 - Nausea and vomiting of clear fluid are common.

Treatment

- Mild water excess requires water restriction only.
- Severe cases
 - Induction of diuresis by mannitol.
 - Careful infusion of small amounts of concentrated (5%) sodium chloride.

Electrolyte imbalance**Sodium depletion (hyponatremia)****Aetiology**

1. Abnormal gastrointestinal tract losses (suction, vomiting and diarrhea). The sodium content of gastrointestinal secretion is nearly similar to that of plasma. The most frequent cause of sodium depletion seen in surgical practice is obstruction of the small bowel, with its rapid loss of gastric, biliary, pancreatic and intestinal secretions by antiperistalsis and ejection, whether by vomiting or aspiration. Duodenal, biliary or intestinal external fistulae are notorious for profound hyponatraemia. Severe diarrhoea causes hyponatraemia with acidosis.
2. Loss of ECF, either externally (burns, marked sweating) or internally as a third space (peritonitis, ascites or ileus). The 'third space' is a collection of ECF that is not functionally available to normal mechanisms maintaining fluid and electrolyte balance. Examples of third spaces are intestinal content in paralytic ileus, tissue edema secondary to trauma or infection and ascites.
3. Excessive urine sodium wastage (diuretics, salt wasting nephritis, and adrenal failure).
4. Blood loss.
5. Restricted dietary intake.
6. Adrenocortical insufficiency results in hyponatraemia with elevated potassium.

Clinical features

The symptoms and signs of sodium depletion are caused by decreased ECF volume.

- The eyes are sunken and the face is drawn, In infants the anterior fontanelle is depressed. The skin is dry and often wrinkled.
- The tongue is coated and dry.
- Unlike the dehydration produced by loss of water only, in water and salt depletion thirst is not particularly in evidence.
- Peripheral veins are contracted.
- Hypovolaemia results in tachycardia, orthostatic hypotension and shock.
- Low CVP.
- Urine is scanty, dark, and of a high specific gravity.

- Haemoconcentration
- Normal or slightly reduced serum sodium. Serum sodium concentration is not a measure of the total body sodium content.

Treatment

1. Restoration of ECF volume by appropriate amounts of sodium-containing fluids such as normal saline (sodium chloride 0.9%) or Ringers lactate.
2. Blood losses should be replaced by blood.

Sodium excess (hypernatraemia)**Causes**

1. If patients are given an excessive amount of 0.9% saline solution intravenously during the early postoperative period, when some degree of sodium retention is to be expected. The result is an overloading of the circulation with salt and its accompanying water.
2. Hyperaldosteronism
 - a. Primary (Conn's syndrome)
 - b. Secondary as in cases of liver cirrhosis
3. Cushing's syndrome.

Clinical features

1. Slight puffiness of the face is the only early sign.
 2. The only reliable clinical sign of total body sodium excess is oedema.
 3. Weight gain parallels accumulation of ECF.
 4. Hypertension.
- Serum sodium concentration is usually normal.

Treatment

Sodium restriction and careful use of diuretics.

Potassium depletion (hypokalaemia)

Since serum potassium content represents a small part of total body potassium, small reductions in its serum level may reflect large body losses of potassium.

Aetiology

1. Excessive vomiting, e.g. pyloric stenosis, intestinal obstruction and paralytic ileus. Prolonged gastroduodenal aspiration with fluid replacement by intravenous isotonic saline solution is a frequent cause of hypokalaemia in surgical patients.
2. External alimentary fistulae.
3. Diarrhoea especially when severe as in cholera, ulcerative colitis, villous tumours of the rectum, or ileostomy dysfunction.
4. Certain types of diuretics as furosemide.
5. Alkalosis due to shift of potassium into the cells without a change in the actual body potassium content.
6. Hyperaldosteronism. Aldosterone causes potassium loss.

Consequences

- Hypokalaemia raises membrane excitation potentials, making nerves and muscles less excitable.
- Risk of supraventricular arrhythmias.
- Potassium depleted patients are prone to develop hepatic coma if they have liver disease.

Clinical features

- Most patients are asymptomatic.

- Early signs of potassium depletion are vague; malaise, and weakness. The speech is slow and slurred.
- Paralytic ileus and distention are seen in some hypokalaemic patients.
- Muscular paresis appears only with extreme depletion. Weakness of the respiratory muscles leads to inadequate ventilation and atelectasis.
- ECG reveals prolonged QT interval, depression of the ST segment, and a lowering or inversion of the T wave.

Treatment

- At a normal pH in the adult, the potassium deficit is calculated as follows $(4.5 - \text{serum potassium concentration}) \times 100$.
- The required quantity of potassium is added to the infusion and distributed all over the day. It should be emphasized that potassium can be very dangerous because hyperkalaemia causes cardiac arrhythmias and asystole. It should never be injected as a bolus, Safe rules for giving potassium are
 - Urine output at least 40 ml/hour.
 - Not more than 40 mmol added to 1 litre.
 - No faster than 40 mmol/hour.

Potassium excess (hyperkalaemia)

Causes

1. Life-threatening potassium excess usually occurs only in association with renal failure and is made worse by tissue destruction or by depletion of sodium or calcium.
2. Acidosis due to shift of potassium outside the cells to the ECF Diabetic patients in whom there is reduced or absent insulin secretion are more prone to hyperkalaemia.

Consequences

- Elevation of serum K to about 5 mmol/L stimulates secretion of aldosterone, which enhances excretion of potassium by the kidney.
- Hyperkalaemia decreases membrane excitation potentials, making cells more excitable. If the serum potassium exceeds 7 mmol/L, intracardiac conduction is slowed and arrhythmia, bradycardia and hypotension may be followed by cardiac arrest.
- The ECG is a sensitive investigation for hyperkalaemia It shows a wide QRS complex and peaked T waves.

Treatment

1. IV calcium gluconate. Calcium antagonizes potassium.
2. IV sodium bicarbonate. Alkalinization encourages intracellular shift of potassium.
3. Dextrose and insulin infusion. 10 units of regular insulin plus 20 gms of glucose. Insulin stimulates deposition of potassium with glycogen. Dextrose is added to prevent hypoglycemia.
4. If the previous measures fail, they should be followed by ion exchange resins, e.g. sodium polystyrene sulfonate, 50 gm in 70% sorbitol by mouth or enema. Repeated enemas may be performed.
5. If all the previous measures fail to lower the serum potassium, dialysis should be started.

Calcium imbalance

Hypocalcaemia may be transient and latent, e.g., hypoparathyroidism following thyroid surgery. It is evidenced by circumoral tingling and numbness, and a positive Chvostek's sign is elicited.

Symptomatic hypocalcaemia develops in established permanent hypoparathyroidism, acute pancreatitis and acute alkalosis, e.g., hyperventilation. The clinical manifestations are neuromuscular hyperactive deep tendon reflexes, muscle and abdominal cramps, carpopedal spasm and rarely convulsions. ECG shows prolonged QT interval.

Treatment is directed to correct the underlying cause. Intravenous calcium gluconate or calcium chloride is given for the acute problem. Chronic hypocalcaemia is treated by vitamin D, oral calcium supplements and aluminum hydroxide gels to bind phosphate in the intestine.

Acid-base imbalance

Metabolic causes of acid-base disturbances are indicated by changes in the standard bicarbonate level and base excess or deficit. Respiratory causes of acid-base disturbances are indicated by changes in the PCO_2 and PO_2 .

Metabolic acidosis

This is a condition where there is a deficit of base or an excess of any acid other than H_2CO_3 .

Aetiology

1. Overproduction of organic acids
 - a. Diabetic ketoacidosis.
 - b. Lactic acidosis of sepsis and shock.
2. Renal failure (acute and chronic)
3. Excessive loss of bicarbonate
 - a. Diarrhea
 - b. Pancreatic or small intestinal fistula.
 - c. Ureterosigmoidostomy.

Pathology

The standard bicarbonate level is lowered and there is a base deficit.

Compensation

- Respiratory. Metabolic acidosis almost always is at least partially compensated by stimulation of respiratory activity. This stimulation results in washing out the CO_2 resulting in a decrease in PCO_2 bringing the HCO_3^-/PCO_2 ratio and the pH back toward normal.
- Renal. Later, renal compensation occurs. Renal excretion of acid increases. This effect is important in chronically acidotic patients with good renal function.

Clinical features

Increased rate and depth of breathing (Kussmaul's respiration).

Treatment

- Mild to moderate acidosis. Therapy should be directed at the underlying cause, e.g., restoration of adequate tissue perfusion.

- More severe acidosis (pH 7.3 or serum bicarbonate below 15 mEq/litre). IV bicarbonate. The required amount of bicarbonate = Body weight (Kg) X 0.3 X base deficit.

Metabolic alkalosis

^ This means a rise in the pH due to accumulation of HCO_3^- .

Aetiology

1. Gastrointestinal losses of H due vomiting or removal (suction) of gastric secretion. This is the most common cause and is usually seen in patients with pyloric stenosis.
2. H^+ movement into the cells associated with hypokalaemia. Intracellular potassium moves out of the cells to compensate for extracellular hypokalaemia, and H moves intracellularly to preserve electroneutrality. This movement results in extracellular alkalosis and a paradoxical intracellular acidosis. Replacement of potassium reverses the process and corrects alkalosis.
3. Bicarbonate retention
 - a. NaHCO_3 administration.
 - b. Milk-alkali syndrome.

Compensation

Respiratory inhibition raises PCO_2 . Compensation is limited by the normal hypoxic respiratory drive when PO_2 reaches 50 mmHg.

Clinical features

- The most striking feature of severe alkalosis is Cheyne-Stokes' respiration with periods of apnea.
- Tetany occasionally occurs.

Treatment

1. In all instances of metabolic alkalosis caused by loss of gastric juice, replacement of chloride is essential to successful therapy. Administration of saline solution is sufficient therapy in mild metabolic alkalosis without hypokalaemia, since the kidney completes the job of correcting acid-base balance by retaining chloride and excreting sodium along with excess bicarbonate. Concomitant hypokalaemia is treated by IV infusion of potassium.
2. In severe metabolic alkalosis not responding to saline or potassium chloride alone, it may be necessary to give IV ammonium chloride or hydrogen chloride very slowly.
3. Tetany is treated by slow IV administration of 10 ml calcium gluconate.

Respiratory acidosis

A fall in pH associated with a rise in PCO_2 is called respiratory acidosis. Respiratory acidosis is always associated with hypoxia. This combination is life-threatening since the rising PCO_2 eventually results in respiratory depression (CO_2 narcosis), with attendant worsening of the hypoxia. Renal compensatory mechanisms are too slow to affect the outcome significantly.

Aetiology

Hypoventilation

1. Inhibition of respiratory drive
 - a. Drugs as opiates or anaesthetics.
 - b. CNS lesions.

2. Disorders of respiratory muscles or the chest wall
 - a. Muscle weakness as in myasthenia or poliomyelitis.
 - b. Morbid obesity.
 - c. Flail chest.
3. Other disorders of ventilation
 - a. Obstructive pulmonary disease.
 - b. Pulmonary oedema.

Compensation

In acute respiratory acidosis, the serum bicarbonate level may not be elevated since renal compensatory mechanisms have not had enough time to act. In chronic respiratory acidosis serum bicarbonate concentration is elevated.

Clinical features

- Restlessness.
- Cyanosis.
- Hypertension and tachycardia in the immediate postoperative period (postoperative pain also causes hypertension and tachycardia).

Treatment

Mechanical ventilation is usually required.

Respiratory alkalosis

Respiratory alkalosis is a condition where carbon dioxide tension in the arterial blood (PCO_2) is below the normal range and the pH is increased.

Table 3.1 Differences between the various types of acidosis and alkalosis

	Defect	Common causes	Bicarbonate/carbonic acid ratio (20/1)	Compensation
Met acidosis	<ul style="list-style-type: none"> ▪ Retention of fixed acids. ▪ Loss of base HCO_3^- 	<ul style="list-style-type: none"> ▪ Diabetes, uraemia ▪ Increased lactic acid ▪ Diarrhea ▪ Small bowel fistulae 	▪ Reduced	<ul style="list-style-type: none"> ▪ Pulmonary (rapid) increased rate and depth of breathing. ▪ Renal (slow)
Met alkalosis	<ul style="list-style-type: none"> ▪ Loss of fixed acids ▪ Gain of base HCO_3^- 	<ul style="list-style-type: none"> ▪ Vomiting, pyloric stenosis 	▪ Elevated	<ul style="list-style-type: none"> ▪ Pulmonary (rapid), reduced rate and depth of breathing. ▪ Renal (slow)
Res acidosis	<ul style="list-style-type: none"> ▪ Retention of CO_2 (hypo-ventilation) 	<ul style="list-style-type: none"> ▪ Depression of respiratory centre ▪ Obstructive pulmonary disease 	▪ Reduced	<ul style="list-style-type: none"> ▪ Renal retention of HCO_3^-, excretion of H^+ ▪ Chlorides shift into red blood cells
Res alkalosis	<ul style="list-style-type: none"> ▪ Excessive loss of CO_2 (hyper-ventilation) 	<ul style="list-style-type: none"> ▪ Hyperventilation 	▪ Elevated	<ul style="list-style-type: none"> ▪ Renal excretion of HCO_3^- and retention of H^+

Aetiology

It results from hyperventilation and is seen in

1. Hysteria.
2. Hyperpyrexia.
3. In patients who are hyperventilated by a mechanical ventilator.

Compensation

Increased renal excretion of bicarbonate but is usually inadequate.

Clinical features

- In most clinical situations respiratory alkalosis is short-lived and well tolerated. When hyperventilation stops, carbonic acid concentration is rapidly restored, and pH returns to normal.
- Reduced ionized calcium may manifest by paraesthesias in the extremities and carpopedal spasm.
- In severe cases, respiratory arrest follows.

Treatment

- When hysteria is the basis for hyperventilation, the patient is instructed to breath into a paper bag.
- Other situations may call for the addition of small amounts of carbon dioxide to the inspired gas mixture.

Practical applications**Diagnosis of imbalances**

Diagnosis of imbalances and an estimation of their magnitude are based on history, clinical signs, and on laboratory studies.

History, e.g., vomiting, diarrhoea, or excessive sweating.

Clinical signs

- Vital signs, pulse and blood pressure.
- Urine output.
- Body weight daily gain in weight hints to sodium or water excess.
- Tissue turgor.
- Oedema.
- Rhales.
- Central venous pressure (CVP), and pulmonary artery wedge pressure (PAWP).

Laboratory values

1. Haematocrit and hemoglobin estimation reflects haemoconcentration or haemodilution. However, it is dangerous to attribute a clinically significant drop in haematocrit reading to dilution in a postoperative patient. Frequently, such patients are bleeding.
2. Serum sodium.
3. Serum potassium concentration should be correlated with the acid-base status.
4. Arterial blood gases (ABGs). The following information are obtained (Table 3.2)
 - a. Blood pH, which is the most direct and accurate measurement of the state of acid-base balance but does not differentiate a normal state from a compensated abnormality.
 - b. Partial pressures of oxygen and carbon dioxide, PO_2 and PCO_2 .

Table 3.2 Arterial blood gases (ABGs) in different types of acid-base disturbancesPO₂ is the partial pressure of oxygenPCO₂ is the partial pressure of carbon dioxide

	pH	PO ₂	PCO ₂	HCO ₃ ⁻	Base
Normal	7.36-7.44	80-110 mm Hg	36-44 mm Hg	22-26 mmol/L	
Met acidosis	Reduced	Normal	Reduced (compensatory respiratory washout)	Reduced	Deficit
Met alkalosis	Elevated	Reduced	Elevated (compensatory respiratory retention)	Elevated	Excess
Res acidosis	Reduced	Reduced	Elevated	Elevation is delayed (compensatory renal retention)	Excess
Res alkalosis	Elevated	Normal	Reduced	Reduction is delayed (compensatory renal excretion)	Deficit

▪ Bicarbonate (HCO₃) and base excess or deficit.

Table 3.2 shows the ABGs of different acid-base imbalances.

- Urine specific gravity, pH, and electrolyte concentrations, e.g. low urine sodium (<20 mmol/24 h) denotes hypovolaemia and a prerenal cause for an increased blood urea.

Postoperative fluid and electrolyte therapy

In prescribing fluid regimens for postoperative patients, three points are considered

- Basal requirements.
- Pre-existing dehydration, electrolytes loss and acid-base disturbances.
- Continuing abnormal losses over and above the basal requirements.

The daily requirements of water and electrolytes in an adult are

- Water 35 mL/kg
- Sodium 1 mmol/kg
- Potassium 1 mmol/kg

The usual daily postoperative fluids for uncomplicated surgery in an adult

- Three litres of fluids = 6 bottles. Add 200ml per day for each 1°C rise in temperature.
- 500 ml saline (0.9% sodium chloride) provides the daily requirements of sodium and chloride.
- The remaining volume requirement (2.5 L = 5 bottles) is given as 5% dextrose (glucose).
- Potassium supplements are given after 48 hours. Either dextrose is replaced by kadalex which contains 27mmol of potassium/L, or potassium supplements in the form of potassium chloride is added,

Points to remember

- Acidosis leads to hyperkalaemia and vice versa, alkalosis leads to hypokalaemia and vice versa.
- Correction of fluid imbalances should be gradual to avoid fluid overload.
- If in doubt as to the state of hydration of a patient you may give a fluid challenge of 200 ml of fluid with close observation of the change CVP and urine output.
- Resuming oral feeding after surgery tends to correct electrolyte imbalances.
- In many conditions of peritonitis and/or septic shock neutrophils adhere to the vascular endothelium and pour large amounts of O₂-derived free radicals as superoxide which make big holes in the vascular lining via damage of the basement

membranes. Water and plasma albumin leak through these holes into the interstitial space and intestinal lumen making up the third space. Relative hypovolaemia results requiring fluid replacement.

- On giving one litre of normal saline (154 meq/L of each of Na and Cl), it is distributed to the extravascular compartment which is roughly four times as large as the intravascular compartment. So, only 250 ml remain in the intravascular compartment (plasma). Similarly, if you infuse one litre of dextrose 5%, it is distributed to the whole body water space and only 8% of dextrose 5% (80 ml) remain in the plasma.
- A patient under fluid therapy with oliguria. The causes are:
 - Catheter problems
 - Prerenal causes increased specific gravity of urine, sodium in urine is less than 20 mEq/L.
 - Acute renal failure.

Some of the bedside features of electrolyte imbalance

- **Hyponatraemia** Headache, lethargy, confusion, weakness and coma.
- **Hypernatraemia** Irritability, thirst, seizures, weight gain and coma.
- **Hypokalaemia** Nausea, vomiting, ileus, paralysis, respiratory complications and cardiac arrhythmias.
- **Hyperkalaemia** Cramps, nausea, vomiting, and cardiac arrhythmias or arrest.

ACUTE HAEMORRHAGE AND BLOOD TRANSFUSION

Classification of haemorrhage

Acute haemorrhage causes loss of both circulating blood volume and oxygen carrying capacity. The common causes include penetrating and blunt trauma, gastrointestinal bleeding, and obstetrical bleeding. Haemorrhage may be classified according to

Site

1. External. Bleeding is visible as it occurs through skin wounds or from a body orifice as in epistaxis or haematemesis.
2. Internal.
 - a. In body cavities, e.g., haemoperitoneum and haemothorax.
 - b. Interstitial, e.g., fracture haematoma.

Type of disrupted vessel

1. Arterial. Blood is bright red and comes in pulsatile jets.
2. Venous. Blood is dark red and comes in a steady flow.
3. Capillary. Bleeding occurs as diffuse ooze of bright red blood.

Timing in relation to trauma

1. Primary haemorrhage occurs at the time of trauma.
2. Reactionary haemorrhage occurs within 24 hours after trauma. As the blood pressure rises due to correction of hypovolaemia or secondary to post-operative pain, an insecure ligature slips or a clot is dislodged.
3. Secondary haemorrhage occurs one to two weeks after trauma due to infection eroding a vessel wall, e.g. after haemorrhoidectomy. It can be fatal if a large artery is involved, e.g., the carotid after sloughing of the skin flaps of a radical neck dissection.

Aetiology

1. Traumatic
 - a. Accidental,

CHAPTER CONTENTS

- Classification of haemorrhage
- Pathophysiological response to haemorrhage
- Clinical picture of haemorrhage
- Management of haemorrhage
- Blood transfusion
 - Collection & storage of blood
 - Blood products
 - Complications
 - Alternatives to homologous blood transfusion

Haemorrhage is classified according to:

- Site External or internal.
- Type of disrupted vessel Arterial, venous or capillary.
- Timing Primary, reactionary or secondary.
- Aetiology Traumatic, pathological or spontaneous.

Common causes of severe haemorrhage

- Trauma Splenic or liver injury
 Hemothorax
 Pelvis fracture
- Major surgery
- Bleeding oesophageal varices
- Bleeding duodenal ulcer
- Ruptured aortic aneurysm
- Pre and postpartum haemorrhage
- Ruptured ectopic pregnancy.

Body response aims to

- Stop bleeding
- Maintain effective blood volume.
- This response is based on neural and endocrinal mechanisms.

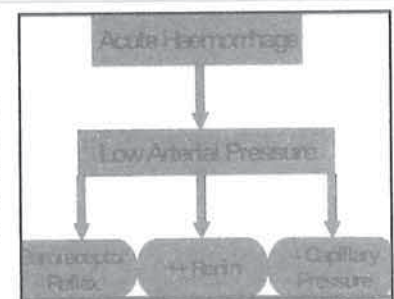


Fig. (4.1) Main physiological consequences of haemorrhage

- b. Surgical.
- c. Interventional procedures, e.g. percutaneous transhepatic cholangiography (PTC).
2. Pathological
 - a. Atherosclerotic (ruptured aortic aneurysm).
 - b. Inflammatory (bleeding peptic ulcer).
 - c. Neoplastic (haematuria in renal cancer).
3. Bleeding diathesis can increase the amount of traumatic and pathological bleeding, or cause bleeding with little or no trauma (spontaneous haemorrhage).

Physiological response to haemorrhage

The physiological response to haemorrhage has two aims:

1. Stopping the bleeding

- Vasoconstriction and retraction of the intima of the injured vessel.
- Platelet plug.
- Blood clotting.

These mechanisms occur in sequence. They are more effective when the vessel is completely transected than when there is a side tear, and in traumatic than in pathological cases when constriction of the vessel may be hindered by inflammatory or degenerative changes in the vessel wall or in its surroundings, e.g., the fibrous tissue in the base of a bleeding chronic peptic ulcer.

2. **Maintaining effective circulatory volume** and perfusion of critical tissues (brain and heart), at the expense of less critical tissues (skin, skeletal muscle and splanchnic area). This is achieved by neural and endocrine factors (Fig 4.1 & 4.2).

- A. **Neural factors.** A sympathoadrenal discharge develops due to decrease in stimulation of arterial baroreceptors (aortic arch and carotid sinus) and atrial stretch receptors leading to reduction of the normal inhibitory discharge in the vagus and glossopharyngeal nerves on the vasomotor centre with consequent stimulation of the sympathetic system. The effects include

1. Constriction of veins, which normally contain two-thirds of the blood volume, displaces blood from the capacitance side of the circulation into the heart.

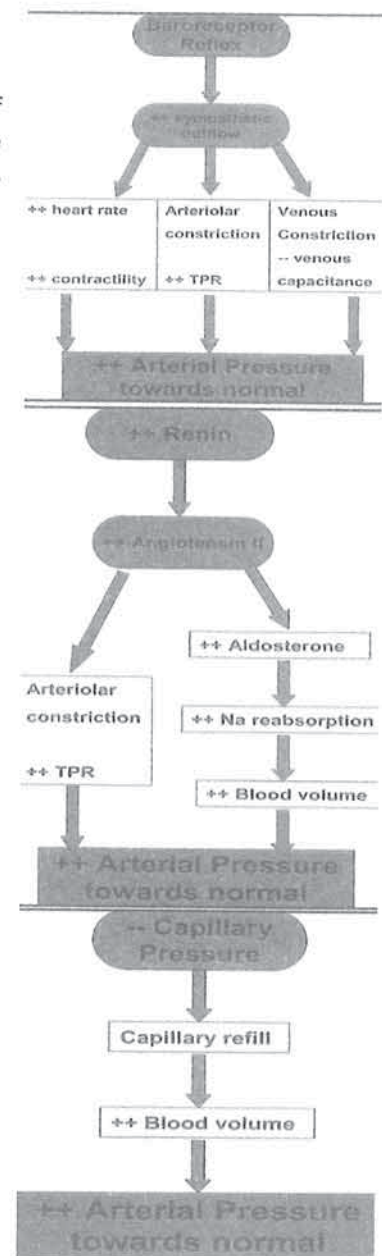


Fig. (4.2) Main compensatory mechanisms for haemorrhage

2. Constriction of arterioles raises the peripheral resistance but this is not uniform. It involves mainly the arterioles of the skin, skeletal muscle, and splanchnic area. Perfusion of the heart and brain is maintained because their metabolic needs override the alpha adrenergic vasoconstrictor discharge.
3. Increased rate and strength of cardiac contraction.

B. Endocrine factors

1. **Catecholamine** discharge occurs from the adrenal medulla and from the nerve endings throughout the autonomic nervous system. They increase the heart rate and myocardial contraction and cause constriction of the arterioles of the skin, kidney and viscera.
 2. The metabolic hormones ACTH, cortisol, growth hormone and glucagon are increased. Insulin release is inhibited by adrenaline and noradrenaline.
 3. The renin-angiotensin aldosterone system. The juxtaglomerular cells of the afferent renal arterioles secrete renin in response to renal hypoperfusion. Renin splits angiotensinogen to angiotensin I which is converted to angiotensin II by a converting enzyme in the lung. Angiotensin II is a powerful vasoconstrictor and stimulates sodium and water retention by a direct action on the kidney as well as indirectly through release of aldosterone from the zona glomerulosa of the adrenal cortex. Angiotensin mediated vasoconstriction takes some 20 minutes to occur, whereas baroreceptor-vasoconstriction occurs within seconds.
 4. **ADH (vasopressin)**. Blood loss greater than 10% stimulates ADH release. ADH increases the permeability of the renal collecting tubules allowing water absorption into the hypertonic renal medullary interstitium. With severe haemorrhage high levels of ADH also cause vasoconstriction.
- C. **Transcapillary refill**. Reduction of blood volume and constriction of arterioles causes a fall in capillary hydrostatic pressure and promotes movement of fluid from the interstitium into the capillaries. The resulting haemodilution increases the blood volume and lowers its viscosity, thus improving effective circulatory volume.

Clinical Picture

The manifestations depend upon

- Amount of haemorrhage
- Rate of haemorrhage
- Cardiovascular reserve

Thus loss of only 500 ml of blood may cause hypotension in a patient with coronary artery disease, whereas in a healthy young adult, e.g. a soldier, losses greater than 1500 ml may not lower systolic pressure initially. The following are the clinical manifestations of hypovolaemia.

Symptoms

1. Weakness and fainting especially when standing.
2. The patient feels cold and thirsty.

Signs

1. The patient looks tired. With decreasing cerebral perfusion, the mental status may vary from anxious to drowsy but the patient usually remains alert,
2. Pulse and blood pressure. With mild blood loss (less than 500 ml), the pulse and blood pressure may remain normal thanks to the efficient compensatory mechanisms. With more blood loss, tachycardia develops but the blood pressure remains stable. With further blood loss, however, the compensatory mechanisms can no longer maintain the blood pressure and progressive hypotension develops (Table 4.2).
3. Pulse pressure (PP) decreases leading to a thready pulse.
4. Respiratory rate. Tachypnea and air hunger.
5. Temperature. Hypothermia occurs. It predisposes to coagulopathy and should be avoided.
6. Skin becomes pale, cold (vasoconstriction) and clammy with slow capillary refill and collapsed veins.
7. Oliguria results due to diminished renal perfusion.

Estimating blood loss

Blood volume is estimated as 70 ml/Kg in adults and 80 ml/Kg in children. In any patient with haemorrhage, it is of vital importance to have a rough estimate of the amount of blood loss determined from

1. **Clinical data.** Four classes of haemorrhage are recognized based on clinical changes in haemodynamic parameters and indices of tissue perfusion (Table 4.1). This table provides only general guidance.
2. **Type of injury.** The haematoma around a closed fracture of the tibia may contain 500-1500 ml of blood, that around a fractured shaft of femur, 500-2000 ml; that in a fractured pelvis, 2000-3000 ml.
3. **Blood loss** at operation is the sum of the amount in the suction reservoir and the amount mopped up by the swabs, the latter is calculated as the difference in swab weight after and before operation multiplied by a correction factor of 1.5-2 depending on the magnitude of the operation.

Investigations

A blood sample is obtained for

1. Complete blood picture, including haematocrit.
2. Coagulation profile
3. Cross matching.

The initial haematocrit is often normal (RBCs and plasma are lost in the same ratio). Some 4-6 hours later, serial haematocrits will show a reduction. Haemodilution is caused by

- Movement of part of interstitial fluid into the circulation.
- Crystalloid replacement of lost blood.

Table (4.1) Clinical parameters in different classes of haemorrhage

	Class I	Class II	Class III	Class IV
Blood loss (in 70 Kg person)	Up to 15% (750 ml)	15- 30% (750-1500 ml)	30- 40% (1500-2000 ml)	>40% (2000 ml)
Mental status	Normal to anxious	Anxious to restless	Aggressive to drowsy	Drowsy to unconscious
Skin	Normal	Pale and cold	Pale and colder	Pale and very cold
Capillary refill	Normal	>2 sec.	>2 sec	>2 sec undetectable
Pulse/minute	<100	100-120	100-140	>140
Blood pressure				
- Systolic	Normal	Normal (supine)	Low	Low
- Diastolic	Normal	Raised	Low	Low
- Pulse pressure	Normal	Low	Low	Low
Respiratory rate	14 -20	20-30	30- 35	>35
Urine (ml/h)	>30	20-30	10-20	0-10

Management of Haemorrhage

1. **Stop haemorrhage.** First-aid treatment is by packing, pressure, and position. A skin wound is covered by a dressing, and pressure is applied manually, by a sphygmomanometer cuff or by a bandage. Tourniquets are contraindicated because of complications unless the limb is going to be amputated. Elevation of the limb above the heart level stops venous and decreases arterial bleeding. Other examples of first aid treatment include the pneumatic anti-shock garment (PASG), which can tamponade lower limb, pelvic and abdominal haemorrhage. The garment increases peripheral resistance and so raises the blood pressure. Another example is balloon tamponade of haemorrhage from oesophageal varices. The definitive management depends upon the cause of bleeding.
2. **Restore blood volume.** Prompt treatment with adequate volume replacement is the keystone of uncomplicated survival. A large bore cannula is inserted in a large peripheral vein, preferably in the upper limb, or by a cut down on the long saphenous vein, if necessary. Volume replacement depends on the class of the haemorrhage.

Class II

- The deficit is estimated at 15-30% (750-1500 ml/70 kg).
- The replacement solution is lactated Ringer's.
- The amount is 3 times the estimated deficit (-3 L). The 3:1 rule serves to replenish the interstitial fluid volume when the crystalloid diffuses out of the capillaries.
- Administration. Two litres are given as a bolus and the response is monitored. If there is definite improvement the remaining litre is given more slowly followed by the maintenance requirements and continued observations (a haematocrit <30 requires blood transfusion). If there is moderate improvement, the possibilities are inadequate replacement, continuing haemorrhage or myocardial insufficiency. Cardiac tamponade and tension pneumothorax must be excluded. Blood transfusion is started if bleeding is still active.

Class III and IV

- The management is as for class II but these patients need blood transfusion. The initial volume of transfused blood is that of the estimated deficit (1500-2000 mL).
- Failure to improve and a rising CVP indicate tension pneumothorax, cardiac tamponade, or cardiac failure. If these are excluded and the patient does not improve, major thoracic, abdominal, or pelvic injury is usually present and calls for immediate operation to control the bleeding. Clean blood in the thorax or

- abdomen can be aspirated, anticoagulated and then transfused back into the patient (autotransfusion), if a cell-saver is available.
- Transfusion should continue until the haematocrit has reached 30%, the urine output 50 ml/hour, and the CVP has risen to the upper half of the normal range.
3. **Optimize oxygen delivery.** Forty percent oxygen is given for class II haemorrhage and 100% for classes III and IV. Intubation and mechanical ventilation and the use of inotropes (dopamine and dobutamine), vasodilators and vasopressors may be necessary if raising oxygen carrying capacity by blood transfusion does not improve oxygen delivery.
 4. **Monitoring** is important to prevent the clinical sequelae and complications of hypovolaemic shock, i.e., cardiac arrest, adult respiratory distress syndrome (ARDS), acute renal failure, GIT dysfunction with stress bleeding, and disseminated intravascular coagulation (DIC).

What to monitor?

- The parameters in Table 4.1 suffice if the patient is fit and responds quickly to bolus infusion of 2 litres of Ringers lactate solution. A Foley catheter is necessary to monitor urine output. Serial rectal temperatures, haematocrits and cardiac monitoring (ECG for early detection of shock-induced arrhythmias) are important.
- Invasive monitoring is needed if the patient is old or has compromised cardiac reserve, class III or IV haemorrhage, or does not respond well to initial crystalloid administration. Central venous pressure (CVP), blood gases, and blood lactate (index of anaerobic metabolism), are necessary. A pulmonary artery catheter is required in complex situations to monitor the function of the left side of the heart.

N.B.

Failure to respond to fluid resuscitation is due to

- i. Continuous bleeding (C.V.P. is low) the causes of massive bleeding include intrapleural – intraperitoneal – retroperitoneal and peripheral bleeding
- ii. Tension pneumothorax – cardiac tamponade – cardiac contusion.

In the three problems C.V.P. is high

Transfusion of blood and its components

Blood is a precious commodity. Its only source is the human being, who willingly parts with some of his life fluid to save the life of a fellow human. Handling of blood should, therefore, be conducted with the deserved dignity, and it should also be administered in its proper indication.

Advances in technology allowed the evolution of synthetic blood substitutes that have the ability to carry oxygen. Though still not the perfect substitute, with further refinement these substances are expected to solve the problem of limited sources and complications of blood transfusion.

Collection and storage of blood

For standard transfusion blood is collected into a citrate anticoagulant solution which also contains dextrose to preserve the viability of RBC's during storage. The simplest solution is acid citrate dextrose (ACD). A better anticoagulant is citrate phosphate dextrose (CPD) or citrate phosphate dextrose plus adenine.

One blood bag (unit) contains 70-100 ml of the anticoagulant and 400-450 ml of blood. Blood must be kept at 2-6°C during storage.

Frozen blood Plasma is removed from the freshly collected blood, then glycerol is added to the red cells. Freezing at degrees of (-80 to -190°C) allows the blood to be kept for long periods. The frozen blood does not have coagulation factors, platelets nor white cells.

Blood products

1. Packed red cells (red cell concentrate). The transfusion of packed red cells is very useful in anaemic patients, in the elderly and in cardiac patients as it improves the oxygenation ability without overloading the circulation.
2. Fresh plasma. This is the component of the blood remaining after packed red cells are separated; It is rich in platelets and coagulation factors.
3. Fresh frozen plasma. Plasma removed from fresh blood is rapidly frozen and stored at -40°C. It is a good source of all the coagulation factors. It is useful to correct coagulation disorders in haemophilia, Christmas disease and in liver failure.
4. Platelets concentrates. The amount obtained from one unit of blood will increase the circulating number of platelets by 10,000 to 15,000/uL. As the half life of platelets is short, they should be freshly prepared. They are very useful in patients with thrombocytopenia.
5. Cryoprecipitate. This is prepared from fresh frozen plasma and is very rich in factor VIII and fibrinogen. It is stored at -40°C.

Table (4.2) Changes that occur during blood storage

	0 days	7 days	14 days	21 days
% of red cell viability	95%	90%	85%	75%
% of platelet viability	95%	0%	0%	0%
% of coagulation factor V and XIII	95%	30%	30%	30%
Potassium content (mmol/L)	3.5	10	25	30

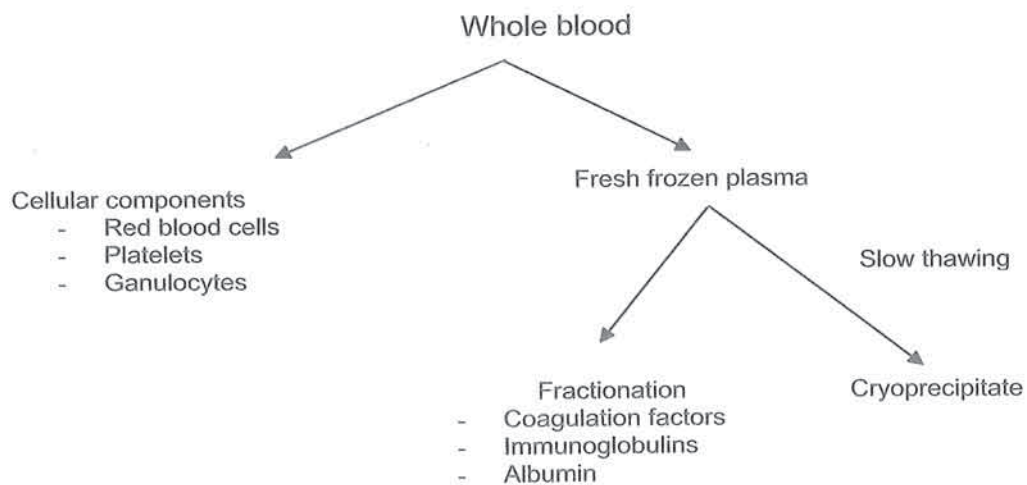


Fig. (4.3): The blood components

Complications of blood transfusion

1. Immunological complications

- Incompatible red cells → Acute haemolytic reaction
- Incompatible white cells → Pyrogenic reaction
- Incompatible platelets → Purpura
- Reaction to a protein in the plasma → Allergic reaction

- a. **Acute haemolytic reaction** Most often these reactions are due to the presence of antibodies in the recipient's blood against one or more of the antigens of the donor's red cells, Occasionally transfused plasma contains a high titre of antibodies against the recipient's RBCs. Clinically haemolytic reactions present after the transfusion of less than 50 ml by fever, chills, constricting pain in the chest, dyspnoea and pain in the flanks. Examination reveals tachycardia and hypotension. In anaesthetized patients the only manifestations of haemolytic reactions are sudden tachycardia, hypotension and bleeding tendency. A major haemolytic reaction will lead to haemoglobinuria, jaundice and acute renal failure due to acute tubular necrosis. Consumption coagulopathy will lead to generalized bleeding tendency.

Delayed haemolytic reaction may occur 5-10 days after transfusion in patients who have been immunized to a foreign antigen by a previous transfusion or pregnancy. It presents by unexplained pyrexia or jaundice.

Management

- Stop the transfusion immediately.
 - Send the donor's blood and a sample of the patient's blood for repeat typing and matching.
 - Correct shock by infusion of crystalloid solution (Lactated Ringer) and IV corticosteroids.
 - Insert a Foley catheter and check that there is an adequate urine output. An osmotic diuretic as mannitol may be needed. Keep an alkaline urine to protect against acute renal failure. IV infusion of sodium bicarbonate may be indicated.
 - Should the patient develop acute renal failure, he must receive the appropriate treatment.
- b. **Pyrogenic reactions.** These are the commonest unpleasant consequences of blood transfusion. The patient develops chills, fever, headache, nausea and vomiting. These reactions are due to the presence of recipient antibodies against some components of the donor's white blood cells. Transfusion is stopped and the patient is given IV aspirin or paracetamol.
- c. Post-transfusion **purpura** may develop in patients who have been previously sensitized to a foreign platelet antigen.
- d. **Allergic reactions** may develop due to immunoglobulin antibody in the recipient complexing with a protein present the donor's plasma. These range from mild itching and urticaria to a severe reaction with laryngeal

edema and collapse. These reactions are common in those patients who received many transfusions in the past. Treatment is by antihistaminics and corticosteroids. If the reaction is severe, blood transfusion should be stopped.

2. **Congestive cardiac failure.** This is liable to occur in elderly persons especially if a large volume of blood is administered too rapidly. It is recommended to transfuse packed red cells rather than whole blood to correct anaemia in elderly persons.

3. Transmission of infection

- Viral hepatitis (B or C) This is now the most feared complication. The virus can be transmitted by whole blood or blood products. It is obligatory to test donors for hepatitis viruses.
 - AIDS/HIV infection can be transmitted by blood or by its products.
 - Syphilis This is now rare. Spirochaetes cannot survive at the blood bank temperature for more than 4 days.
 - Malaria The disease is transmitted only by red cells, not by blood components.
 - Septicaemia Bacteria can survive, but they cannot multiply significantly in refrigerated blood. However, if the blood is allowed to warm, bacteria can grow and Gram-negative endotoxins can cause septicaemic shock.
4. **Hyperkalaemia** With prolonged storage of blood, there is progressive loss of potassium from erythrocytes into the plasma. Transfusion of several units of aged blood may produce cardiac arrhythmias or even arrest due to hyperkalaemia.
 5. **Citrate intoxication** Excess citrate will bind to the recipient's calcium leading to hypocalcaemia which augments the effects of hyperkalaemia on the myocardium. If more than 2 units of blood are administered, it is important to administer 10 ml of 10% calcium gluconate for each two units of blood.
 6. **Air embolism.**
 7. **Transfusion related acute lung injury (TRALI)** This is the result of incompatibility between donor's antibodies and recipient granulocytes. It gives a clinical picture similar to ARDS.
 8. **Complications of massive blood transfusion** This implies transfusion of 2500 ml of blood at one time or 5000 ml or more over 24 hours.
 - Hypothermia. A special warming unit should be used to warm the blood before transfusion as hypothermia can cause acidosis or cardiac arrest.
 - Hyperkalaemia.
 - Hypocalcaemia.
 - Coagulation failure. This is due to the dilution of clotting factors and platelets when large volumes of stored blood are being used to replace blood losses, because stored blood is poor in platelets, factor VIII and factor V. In these situations it is recommended to transfuse one unit of fresh frozen plasma and platelets for every unit of stored blood.
 - Diminished O₂ carrying capacity of red blood corpuscles.

Alternatives to homologous blood transfusion

1. Autologous blood transfusion A patient who is going to have a major elective operation, can donate some units of his own blood over several days. The blood is kept in the refrigerator to be given back to him during surgery.
2. Preservation of the blood lost during surgery and its reinfusion to the patient. This needs a special apparatus (cell saver).

Remember

- Hypotension in the supine position in a fit patient means a loss of at least 1500 ml of blood.
- It is no longer recommended to transfuse fresh blood to correct coagulopathies. The specific deficient component should be used.

Table (4.3): Indications for transfusion of blood and its products

Product	Indication	Precautions	Storage life
Whole blood	Class III & IV haemorrhage	ABC & Rh	21 days
Red cell concentrates	Severe anaemia	ABC & Rh	21 days
Fresh frozen plasma	Bleeding due to non-specified coagulation factor deficiency Coumarin overdose	ABO	1 year at -40°C
Platelet concentrates	Primary or secondary thrombocytopenia	ABO	24 - 72 hours
Cryoprecipitates of factor VIII & fibrinogen	Bleeding with fibrinogen depletion	ABO	1 year at -40°C
Factor VIII	Haemophilia A		2 years
Factor IX	Haemophilia B. Coumarin overdose		
Albumin 5% or 20%	Acute volume expansion. Hypoalbuminaemia		4 years

HAEMOSTASIS

Introduction

Haemostasis is the physiological arrest of bleeding. It involves vasoconstriction, platelet plug formation and fibrin deposition to form a stable clot sealing the disruption in the vessel wall. A fourth mechanism, fibrinolysis breaks down the clot, restoring vascular patency after bleeding has stopped. Natural haemostasis sometimes fails leading to excessive bleeding or thrombosis.

CHAPTER CONTENTS

- Introduction
- Natural haemostasis
 - Primary haemostasis
 - Secondary haemostasis
 - Fibrinolysis
- Haemostatic disorders
 - Congenital disorders
 - Acquired disorders
- Preoperative evaluation of haemostasis
- Excessive operative and postoperative bleeding
- Surgical haemostasis

Natural haemostasis

Primary haemostasis

Arrest of haemorrhage within the first few minutes is achieved by

1. Vasoconstriction of the disrupted vessels. This is achieved by
 - Contractile proteins in the endothelium (even capillaries constrict).
 - Neurogenic reflexes.
 - Direct smooth muscle stimulation (spasm).
 - Vasoconstrictors, e.g. thromboxane.
 - Vasoconstriction is more efficient in arteries than veins because arteries contain more muscular fibres. Vasoconstriction is less effective in the following conditions
 - When a vessel is partially injured.
 - In stiff atherosclerotic vessels.
 - In large veins (poor muscle coat).
2. Platelet plug. Platelets serve the following haemostatic functions
 - Adhere to exposed subendothelial collagen.
 - Release the contents of their granules, e.g. ADP, and serotonin.
 - Aggregate to form a plug that seals the bleeding vessel. Thromboxane from platelet membrane phospholipid, ADP, serotonin, thrombin etc, are responsible for aggregation.
 - Platelet phospholipid has a vital role in blood coagulation.
3. Tamponade of the bleeding by surrounding tissue tension. Interstitial haemorrhage tends to be less profuse than external or internal haemorrhage.

Secondary haemostasis

Maintenance of haemostasis after the first few minutes requires that the platelet plug be reinforced by fibrin deposition, i.e. coagulation. Coagulation is activated by two mechanisms (Fig. 5.1).

1. **Extrinsic**; initiated by activation of factor VII upon admixture of plasma and tissue factor.
2. **Intrinsic**, initiated by activation of factor XII upon contact with a non-endothelial surface, more directly by activation of factor IX through activated factor VII.

Sequential activation occurs locally; any activated coagulation factor spilling into the general circulation is neutralized by inhibitors such as antithrombin III and proteins C and S. Deficiency of these proteins may cause spontaneous thrombosis, e.g. mesenteric vascular occlusion.

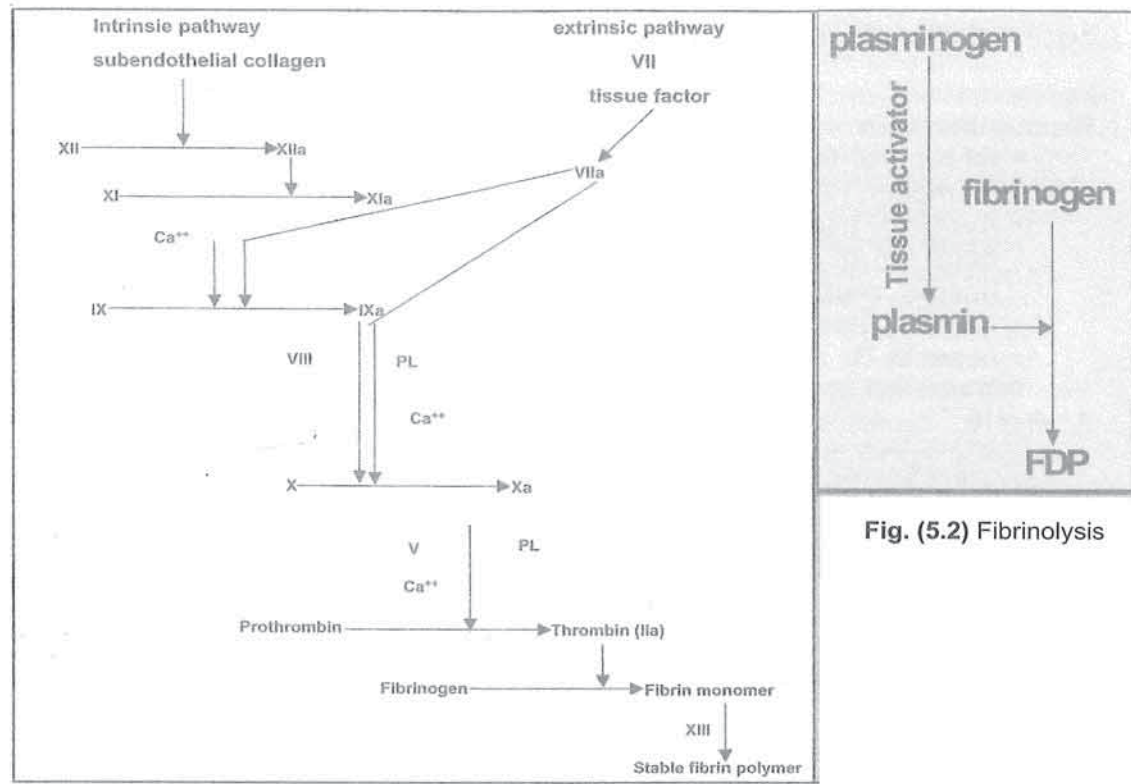


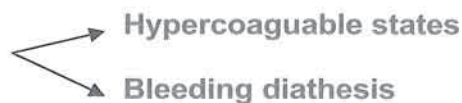
Fig. (5.1) Coagulation mechanism
a (Activated factor) PL (Platelet phospholipid).

Fig. (5.2) Fibrinolysis

Fibrinolysis

Once haemostasis is achieved, the clot obstructing the vessel is no longer needed. Circulating plasminogen and tissue plasminogen activator bind to the fibrin clot leading to local formation of plasmin which digests the clot and restores vascular patency (Fig. 5.2). Circulating inhibitors neutralize plasminogen activators and plasmin which form or overflow into the circulation. The products of fibrin digestion by plasmin are called fibrin degradation products (FDP). D-dimers result from plasmin cleavage of cross linked fibrin and constitute a marker of fibrinolysis.

Haemostasis disorders



Hypercoagulable States

An acquired tendency to thrombosis occurs in pregnancy, estrogen use (HRT or contraception), and malignancy. This tendency is increased if there is an associated inherited thrombophilia, e.g. deficiency of a natural anticoagulant (antithrombin III, protein C or protein S).

Haematology consultation is required for a personal or family history of spontaneous/recurrent thrombosis whether arterial and/or venous as well as obstetric history of recurrent miscarriages. Surgical patients should have standard DVT prophylaxis. Estrogen should be stopped 1 month before elective surgery.

Bleeding diathesis Congenital or acquired

Congenital disorders

1. **Haemophilia A and B** These are due to deficiency of factors VIII and IX respectively. Haemophilia A is the most common congenital coagulopathy. Inheritance (in both) is

sex-linked (from females to males). Manifestations vary from frequent episodes of spontaneous bleeding starting in childhood, (<1% of factor activity). to bleeding only after trauma or surgery (factor level 5-20% of normal). Patients are liable to recurrent haemarthrosis.

Management involves

- Infusion of factor concentrate within 1 hour before surgery and for 10 days thereafter.
 - Avoiding aspirin and the nonsteroidal anti-inflammatory drugs (NSAIDs) because of their antiplatelets effect, and avoiding IM injections (large haematomas can develop).
 - As the patient will need repeated transfusion, he needs vaccination against hepatitis B.
2. **Von Willebrand disease (vWD)** von Willebrand disease, together with haemophilia A and B, constitute more than 95% of inherited coagulopathies. Inheritance is autosomal dominant with variable expression. vWD is due to deficiency of vW factor which enhances platelet adhesion and acts as a carrier to factor VIII, preventing its premature destruction. Post-surgical bleeding can be alleviated by infusion of intermediate-purity factor VIII concentrate, (which contains both factor VIII and vW factor).

Acquired disorders

These are more prevalent than congenital ones.

1. Hepatic disorders

Both acute and chronic liver diseases may be accompanied by haemostatic abnormalities.

Aetiology is multifactorial.

▪ **Coagulation factors**

- Decreased concentration of all clotting factors except factor VIII (synthesized in other organs).
- Dysfibrinogenaemia. Defective polymerization of the fibrin clot.
- Antithrombin III level is decreased, this contributes to intravascular coagulation in cirrhotics.

Prothrombin time (PT) is the first to be prolonged (decreased synthesis of factor VII) which has a short half life. In more severe diseases, partial thromboplastin time (PTT) is also prolonged (decrease synthesis of other factors, e.g. V, IX and X).

▪ **Platelets**

- Thrombocytopenia due to splenic sequestration or destruction due to hypersplenism.
- Abnormal function due to preponderance of small, less active platelets.

The bleeding time is, however, normal in most cirrhotics.

▪ **Fibrinolysis**

- Reduced synthesis of inhibitors of fibrinolysis, e.g., alpha 2 anti-prothrombin.
- Impaired clearance of plasminogen activators. Fibrin degradation products (FDP) level may increase.

▪ **Treatment**

- Vitamin K administration.
- In the setting of bleeding or an invasive procedure give
 - Fresh frozen plasma (FFP) if PT or aPTTT >1.5 times normal.
 - Cryoprecipitate to correct severe hypofibrinogenemia (<100 mg/dl).

- Desmopressin (0.3 ug/kg) can raise the levels of factor VIII and vWF, and shorten the bleeding time before an invasive procedure.
- Tranexamic acid or other fibrinolysis inhibitors may be useful in upper GIT haemorrhage.

2. Vitamin K deficiency

The sources of this fat soluble vitamin are the diet and bacterial synthesis in the colon. Bile is necessary for its absorption. Causes of deficiency include

- Inadequate diet (or TPN).
- In debilitated patients given prolonged broad spectrum antibiotics (reduce colonic bacteria).
- Cholestatic jaundice.
- Malabsorption.
- Oral anticoagulants.

Vitamin K is a co-factor in the synthesis of factor II, VII, IX, and X. Deficiency leads to easy bruising and increased traumatic bleeding. Both the PT and PTT are prolonged. Treatment is with vitamin K₁ by slow IV infusion (5-10 mg), or daily IM injections for 3 days (10-20 mg/day). In an emergency, factor concentrates, (II, VII, IX and X), or fresh frozen plasma may be needed as well as blood.

3. Disseminated intravascular coagulation (DIC)

Two processes occur.

- Widespread activation of coagulation within the circulation leading to consumption of coagulation factors, including fibrinogen, and to depletion of platelets.
- Increased levels of fibrin degradation products (FDP), which worsen the coagulopathy by inhibiting fibrin polymerization.

Causes include

- Septicaemia.
- Severe shock, trauma, and burns.
- AGO incompatible transfusion.
- Malignancies, e.g., metastatic carcinoma of the lung, pancreas, stomach, prostate, and leukaemia.
- Obstetric accidents (eclampsia, abruptio placenta, amniotic fluid embolism, and retained dead fetus).

Diagnosis is suspected by

- Diffuse bleeding from wounds, incisions, drain and venopuncture sites.
- Widespread bruising, purpura and mucosal bleeding.
- Occasionally, the thrombi are not lysed quickly and ischaemic manifestations occur, e.g., gangrene of skin and digits.
- Uncoagulable blood in severe cases.

Laboratory features include

- Thrombocytopenia.
- Prolongation of PT, PTT and low fibrinogen level (N=2-4 g/L).
- Raised level of FDP and D-dimers.

Treatment

- Treatment of the underlying cause to stop the cycles of coagulation/fibrinolysis, e.g. draining an abscess and antibiotics for infection.
- Replacement of consumed coagulation factors and platelets with fresh frozen plasma (3-4 units initially) and cryoprecipitate when available (it is rich in factor VIII and fibrinogen), and platelet transfusion (10-12 packs may be needed initially).

- Blood transfusion to restore circulating blood volume and oxygen carrying capacity since hypoxia exacerbates DIC.
- Heparin is only used if there is a large vessel thrombosis.

4. Anticoagulants

Anticoagulants are used to prevent or treat arterial or venous thromboembolism. Bleeding may occur if the dose is not properly adjusted. A problem arises when a patient on warfarin requires surgery. Preoperatively the INR must be allowed to drop below 1.5 before the surgery to avoid excessive bleeding. This is achieved by stopping the daily warfarin dose for 4-5 days before elective surgery or using FFP in an emergency (or recombinant factor VIIa to avoid risk of blood component therapy).

An INR <1.5 must be confirmed before surgery.

Preoperative heparin is required if stopping warfarin is associated with a high risk of recurrent thrombosis. LMWH, e.g. enoxyparin, 1 mg/Kg SC every 12 hours is started 36 hours after the last dose of warfarin and stopped 24 hours before operation.

Postoperatively LMWH is restarted 24 hours post-procedure. Warfarin is restarted on postoperative day 1 at the preoperative dose. Stop heparin and continue with warfarin when the INR reaches target level, e.g. 2-3.

5. Massive blood transfusion (Chapter 4)

6. Platelets disorders

Thrombocytopenia

- Decreased production by the bone marrow (leukaemia, cancer infiltration, viral infection and chemotherapy).
- Increased destruction (ITP, drug-induced e.g. heparin, thiazides and sulpha).
- Increased sequestration in hypersplenism.

Disorders of platelets' functions

- Drugs as aspirin and NSAIDs inhibit cyclooxygenase and prostaglandin synthesis, thus they interfere with platelet adhesiveness.
 - Dipyridamole (Persantin) reduces platelet adhesiveness.
 - Uraemia and hypothermia can cause platelet dysfunction.
- N.B. Aspirin and NSAID should be stopped 7 days before surgery.

Preoperative evaluation of haemostasis

All patients scheduled for elective surgery should be evaluated for a possible haemostatic defect by history, examination and, when appropriate, laboratory tests.

History

This is the most important part of the haemostatic evaluation. Points to be covered include

1. **Family history of bleeding.** If positive in maternal grandfathers, maternal uncles, or a brother, it suggests haemophilias (A or B); in a parent or sibling of either sex, it suggests vWD (autosomal dominant inheritance with variable expression).
2. **History of bleeding tendency,** e.g., spontaneous bleeding, prolonged bleeding after a haemostatic challenge (circumcision, dental extraction, surgery or trauma), bleeding from multiple sites, or easy bruisability.
3. **Age at onset of bleeding.** A long history and onset in childhood suggest a hereditary disorder. Recent onset in an adult suggests an acquired disorder.

4. **Inquire about liver disease, chronic renal failure, massive blood transfusion and drug intake.**
5. **Characters of the bleeding**
 - Defects of primary haemostasis (platelet/vessel abnormalities). Bleeding is superficial i.e. skin and mucous membranes, such as petichae, purpura, easy bruisability, epistaxis, and menorrhagia. Excessive oozing occurs immediately following surgery or trauma and is usually controlled by local pressure. Visceral bleeding, e.g., CNS is not common.
 - Defects of secondary haemostasis (coagulation/fibrinolysis). Bleeding is deep, e.g. haemoarthrosis, haematomas in muscles, retroperitoneal or visceral, in addition to easy bruisability. Onset following surgery or trauma is delayed and local pressure is usually ineffective. Mucous membrane bleeding is not common.
 - Excessive fibrinolysis or thrombolytic agents cause generalized bleeding, e.g. from the wound, drain site, or IV sites.

Examination

The following points are checked in the preoperative patient if a bleeding tendency is suspected.

1. Cutaneous signs of liver disease, e.g. jaundice, and spider naevi.
2. Skin and mucous membranes are examined for bleeding, petichae, or bruising.
3. Cavernous haemangioma may trap platelets leading to thrombocytopenia.
4. Musculoskeletal system. Muscle haematomas, and haemoarthrosis (coagulopathy).
5. Abdomen. Hepatomegaly, and splenomegaly. The latter may be the cause of platelet sequestration and thrombocytopenia.

Tests of Haemostasis

Indications

1. Patients with a personal history or a family history of abnormal bleeding.
2. Patients with diseases or who receive medications that can interfere with haemostasis.

Tests of primary haemostasis

1. Platelet count. Normal = 150.000-400.000/uL.
 - Spontaneous bleeding occurs with counts 10.000-20.000/uL.
 - Prolonged bleeding with minor trauma occurs with counts 20.000-50.000/uL.
 - Prolonged bleeding with major surgery/trauma occurs with counts 50.000-100.000/uL.
 - No haemostatic abnormality occurs in counts more than 100.000/uL.
2. Bone marrow aspiration and biopsy.
 - Megakaryocytes are normal or increased in thrombocytopenia due to peripheral destruction or sequestration.
 - Megakaryocytes are rare in bone marrow damage, e.g., chemotherapy, or replacement, e.g. leukaemias.
3. Bleeding time (BT) is prolonged with platelet and vascular defects.
4. Tests of platelet function (adhesion, release, aggregation) may be necessary when the BT is prolonged but platelet counts are normal.

Tests of secondary haemostasis

1. Prothrombin time (PT) measures the time of clotting through the extrinsic and common pathways which involve factor VII and factors X, V, II and fibrinogen respectively.
2. Partial thromboplastin time (PTT) measures the time of clotting through the intrinsic pathway (factors XII, XI, IX, and VIII) and the common pathway.

3. Thrombin time (TI) measures the time of clotting after thrombin is added to plasma. It is sensitive to abnormalities of fibrin formation, e.g. due to FDP in DIC.

Other tests

1. Fibrinogen level is decreased in DIC.
2. Fibrin degradation product (FDP) and D-dimer screens for fibrinolysis.
3. Individual coagulation factor assays.
4. Clot stability test evaluates factor XIII, deficiency of which is not detected by the PT or PTT.

Excessive operative and postoperative bleeding

Causes

1. Inadequate surgical haemostasis is the commonest cause. If the bleeding persists, it becomes complicated by DIC and the coagulopathy of massive blood transfusion.
2. Haemorrhagic diathesis not detected preoperatively. The PT, PTT and TT are not very sensitive and may be normal even when the deficient factor concentration is down to 20% of normal level. A PT of 2-3 seconds above normal level is significant.
3. Acquired intraoperative coagulopathy.
 - DIC including incompatible blood transfusion.
 - Massive blood transfusion (thrombocytopenia, depletion of clotting factors, e.g. VIII and V, when blood is not very fresh, and hypothermia if blood is not properly warmed).

Management

1. PT, PTT, and platelet count are ordered.
2. Fresh frozen plasma 3-4 units initially replaces the consumed or diluted clotting factors.
3. Platelet transfusion may be necessary to raise platelet count above 50,000/uL.
4. Correction of hypothermia, and acidosis.
5. Proper surgical haemostasis.

Surgical haemostasis

During surgery bleeding is controlled by one of the following methods.

1. Ligation. A bleeding vessel is clamped by an artery forceps. A ligature is tied around it, then the clamp is released.
2. Under-running suture. This is used if the bleeding point cannot be controlled by an artery forceps.
3. Electrocautery (diathermy) is used to stop bleeding from small blood vessels that are about 3 mm in diameter or less. This is either
 - Monopolar diathermy, where the electric current passes through the patient's body between two electrodes. One electrode (passive) is broad and is usually placed on the lower limb or the back. The other (active electrode) is usually a forceps with a fine tip, and which the surgeon uses to grasp a bleeding point. As the current passes between the electrodes it becomes concentrated at the fine-tipped forceps. This generates heat which causes tissue coagulation.
 - Bipolar diathermy, where the current passes between the two blades of a forceps which the surgeon uses to grasp the bleeding point.
4. Clips
5. Laser
6. Repair of injured large vessels (chapter 14).

- Primary haemostasis is achieved by vasoconstriction, platelet plug formation and tamponade by the surrounding tissue tension.
- Secondary haemostasis is achieved by blood coagulation.
- Disorders of haemostasis may be due to congenital but more commonly acquired disorders.
- Acquired disorders of haemostasis are due to hepatic disorders, vitamin K deficiency, DIC, anticoagulants, massive blood transfusion, or platelets disorders.

SHOCK

Introduction and classification

Shock is a condition that leads to inadequate tissue perfusion that results in impaired cellular metabolism. To maintain an adequate blood flow to the tissues, a fine balance exists between the blood volume, myocardial contractility and the peripheral resistance. A serious disturbance in any of these three components can lead to shock.

Classification (Fig. 6.1)

1. **Hypovolaemic** shock is due to diminished blood volume.
2. **Cardiogenic** shock is due to inefficient myocardial function.
3. **Neurogenic** shock is due to peripheral vasodilatation, reduced peripheral resistance, and peripheral pooling of blood.
4. **Anaphylactic** shock is due to antigen antibody reaction that also leads to peripheral pooling of blood.
5. **Septic** When sepsis is severe enough to stimulate the reticuloendothelial system in an excessive abnormal way, chemical mediators are released. These mediators affect the microcirculation resulting in deficient perfusion of the tissues.
6. **Endocrinal** shock is due to acute deficiency of corticosteroids.
7. **Obstructive** shock This type of shock occurs in patients in whom although the blood volume is normal, there is interference with cardiac filling. It occurs in tension pneumothorax, pericardial tamponade and massive pulmonary embolism.

In some cases a patient may have a combination of two or more of the above-mentioned categories. A trauma victim, for example, may have blood loss from an injured viscus that produces hypovolaemic shock, in addition to blood collection in the pericardium that causes cardiac tamponade and obstructive shock. In another trauma victim hypovolaemic shock may be compounded by the development of neurogenic shock that is caused by cervical spine fracture which severs the spinal cord.

CHAPTER CONTENTS

- Introduction and classification
- Hypovolaemic shock
 - Aetiology
 - Pathophysiology
 - Clinical picture
 - Treatment
- Cardiogenic shock
 - Aetiology
 - Clinical features
 - Treatment
- Neurogenic shock
- Anaphylactic shock
- Septic shock
 - Aetiology
 - Pathophysiology
 - Clinical features
 - Treatment
 - Prognosis
- Obstructive shock

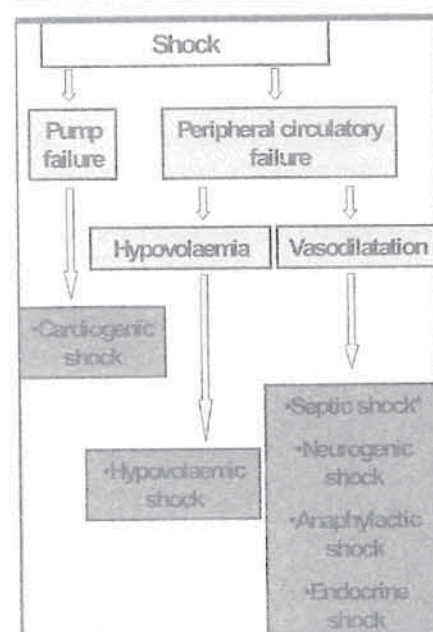


Fig. (6.1) Classification of shock

Common causes of severe haemorrhage

- Trauma
 - Splenic or liver injury
 - Hemothorax
 - Pelvis fracture
- Major surgery
- Bleeding oesophageal varices
- Bleeding duodenal ulcer
- Ruptured aortic aneurysm
- Pre and postpartum haemorrhage
- Ruptured ectopic pregnancy.

Hypovolaemic Shock

Aetiology

This type is due to diminished blood volume which may occur secondary to loss of

1. Blood as in internal or external haemorrhage.
2. Plasma as in burns, acute pancreatitis and peritonitis.
3. Sodium-containing fluids as in severe vomiting, diarrhoea, intestinal obstruction or a high output intestinal fistula.

Pathophysiology

The classical example of hypovolaemic shock is haemorrhage.

The sympatho-adrenal response

The body response to haemorrhage aims to stop the bleeding and to divert blood from the relatively less critical tissues (skin, gastrointestinal tract and the kidney) to the critical organs as the brain and the heart. Both neural and endocrinal factors work together in superb harmony to fulfill this function (Chapter 4).

The clinical picture of a patient with hypovolaemic shock is a reflection of this intense sympathoadrenal response (Chapter 4). If the patient is rapidly treated by adequate volume replacement, vital signs will return to normal and he will be fine. However, with persistent hypovolaemia or lack of prompt treatment, the intense sympathoadrenal response starts to produce harmful effects as follows

Microcirculatory changes (Fig. 6.2)

1. Under the effect of catecholamines the precapillary sphincters constrict. Less blood enters the capillaries and the capillary circulation becomes sluggish.
2. Untreated hypovolaemia thus leads to hypoxia. Under normal conditions only one third of the capillary bed is open at a time. Under ischaemic conditions, however, the body reacts by opening more capillaries. The results are
 - Further slowing of capillary circulation, the so called sludging of blood.
 - Sludging encourages spontaneous coagulation of blood in these capillaries. If extensive, it is called disseminated intravascular coagulation (DIC). This depletes coagulation factors and induces bleeding tendency in the rest of the body (consumption coagulopathy).
3. A sluggish circulation, and DIC compound tissue hypoxia and affect the function of capillary endothelium. The result of the latter is leakage of large protein molecules from the vessels into the interstitial space dragging with them huge amounts of fluid. This third space loss of fluid further reduces the blood volume.

Knowledge of this sequence of events reflects the importance of early restoration of adequate blood volume.

Cellular Derangement

1. Lack of oxygen forces the cells to rely on anaerobic glycolysis for the production of energy and ATP. Anaerobic metabolism produces lactic acid and a small amount of energy,

How much of your 5 litre blood volume can you lose at once?

- 10%. You donated a unit of blood. Thank you.
- 20%. You will probably feel a little sick.
- 40%. You will probably go into hypovolaemic shock.

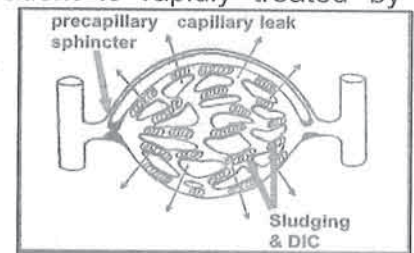


Fig (62): Microcirculatory changes in hypovolaemic shock

- Precapillary sphincters constrict.
- Sludging then DIC.
- Capillary leak.

2. With persistent hypoxia, cellular demand exceeds production of energy. Cellular functions deteriorate. Most important is the failure of Na/K pump which results in inability of the cells to get rid of sodium with consequent progressive accumulation of intracellular water.
3. The end result is rupture of lysosomal and plasma membranes and cell death.
4. Though the vital organs are originally spared as a result of the sympatho-adrenal response, they are eventually exposed to hypoxia. If extensive cell death affects these organs, the result is irreversible shock.

Acid-base imbalance

Shock is accompanied by metabolic acidosis, which is caused by

1. Accumulation of lactic acid as a result of anaerobic metabolism. Initially the body tries to correct acidosis by the buffering system, by hyperventilation, and by renal conservation of bicarbonate.
2. Renal failure from prolonged ischaemia, aggravates acidosis.

Individual organs (Fig. 6.3)

1. **Heart.** Myocardial contractility is impaired by
 - The reduction in coronary arteries perfusion.
 - Direct myocardial depressants as cachectin I tumour necrosis factor (TNF) released from the hypoperfused gut, and by leukotrienes and platelet activating factor that are released in trauma.
2. **Gastrointestinal tract** The mucosa is the most sensitive layer to ischaemia.
 - Ischaemia of the gastric and duodenal mucosa may produce superficial ulcers, the so-called 'stress ulcers' which may bleed.
 - The normal colon mucosa does not allow invasion of the blood stream by its microorganisms. With persistent hypoperfusion of the bowel, there may be translocation of bacteria and endotoxins from the lumen into the circulation. This phenomenon plays a role in septic death in previously, shocked patients even after resuscitation.
3. **Liver** Ischaemic hepatic dysfunction is a frequent component of multiple organ system failure in patients who survive initial therapy.
4. **Kidneys**
 - Total renal blood flow decreases due to the sympathoadrenal response.
 - Intrarenal distribution of blood flow shifts from the cortex to the medulla. Renal renin leads to increased levels of angiotensin II which is a potent vasoconstrictor, releases aldosterone and antidiuretic hormone. The net effect is to decrease glomerular filtration rate and increase tubular reabsorption of salt and water in an attempt to maintain the circulating intravascular volume deficits. Prolonged hypotension ends in acute tubular necrosis (ATN) which is a part of the multiple organ system failure.
5. **Lungs.** See ARDS (Chapter 28).

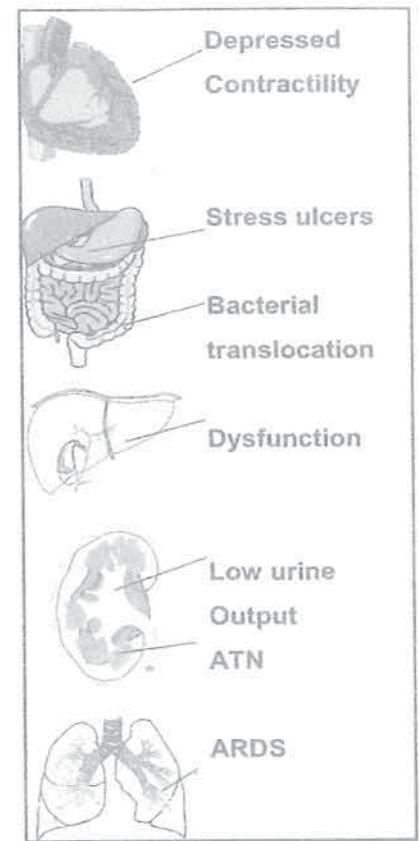


Fig. (6.3) Organ affection in hypovolaemic shock

Clinical Picture (Chapter 4)

Treatment

Treatment of hypovolaemic shock wouldn't be successful without arresting the volume loss. As mentioned in chapter 4 hemostasis is of supreme importance. The mainstays of initial treatment of shock are the infusion of fluids and the administration of oxygen. Close clinical and laboratory monitoring guides the magnitude of resuscitation.

1. Fluid resuscitation.

- **Venous access.** At least two large-gauge catheters are inserted into appropriate veins. At the same time, blood is drawn for typing and cross matching (Chapter 4).
- **Lactated Ringer's solution.** An infusion of lactated Ringer's solution is begun immediately. The lactated Ringer's solution is run at a rapid rate so that in a period of 45 minutes between 1000 and 2000 ml of lactated Ringer's solution are given intravenously. The procedure is a highly effective therapeutic trial to determine the pre-existing amount of blood loss or the presence of continuing blood loss. It is often observed that the blood pressure will return to normal, become stable, and remains so in patients with minimal blood loss and in whom haemorrhage is not continuing.
- **Blood.** If blood loss has been severe or haemorrhage is continuing, the elevation of blood pressure and reduction in pulse rate that occur with rapid IV infusion of lactated Ringer's solution are usually transient. When this occurs, blood that has been accurately typed and cross-matched is given immediately.
- **Colloid solutions.** In the absence of whole blood, many substances have been proposed as human plasma, albumin solution, dextran and artificial blood substitutes. Hypovolaemic shock from other causes other than bleeding, e.g., plasma loss in major burns, or crystalloid loss in intestinal obstruction does not usually need blood, and infusion is by plasma or crystalloids respectively.

HYPOVOLAEMIC SHOCK

Decreased blood volume

Common causes

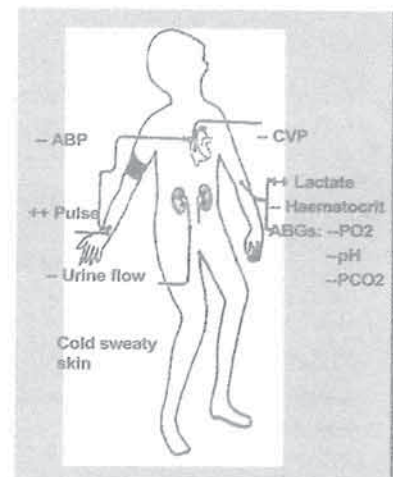
- Hemorrhage
- Burns
- Acute pancreatitis

Early signs

- Tachycardia (mild)
- Orthostatic hypotension
- Anxiety
- Sweating
- Pallor

Late signs

- Depressed mental status
- Decreased BP
- Tachycardia (marked)



The best indicator of tissue perfusion is urine flow.

Lab tests to assess tissue perfusion

- pH
- Lactate

adjustment of rate and concentration depends on arterial gas measurements.

- Evidence of respiratory failure is an indication for endotracheal intubation and mechanical ventilation.
3. **Monitoring.** A patient with hypovolaemic shock should have meticulous monitoring to check the adequacy of volume replacement.
- Clinical parameters as the pulse, blood pressure, state of filling of veins and capillary perfusion.
 - A Foley catheter is introduced to check urine output every hour. Optimum output is 0.5-1 ml/kg/hour.
 - Central venous pressure (CVP). The CVP is the venous pressure in the right atrium and is equal to the end-diastolic pressure in the right ventricle and is a measure of the preload to this chamber. To measure the CVP, a catheter is placed in the right atrium via the median-cubital vein, the subclavian vein or the internal jugular vein. The position of the venous catheter is checked by chest X-ray. The radiograph also serves to rule out pneumothorax due to accidental pleural injury during its insertion. The normal pressure is 5-10 cm of water. Assuming that cardiac function is normal, the CVP roughly corresponds to the blood volume. Thus, a high pressure indicates overtransfusion, while a low pressure indicates hypovolaemia.
 - Pulmonary artery wedge pressure (PAWP). When the right side of the heart is functioning abnormally, it is highly probable that the left side of the heart is equally affected. In such a case, it is recommended to measure PAWP by the use of a Swan-Ganz catheter which is passed into a small branch of the pulmonary artery where it becomes wedged (Fig. 6.4). As the balloon of the catheter occludes this small branch, the pressure measured at the catheter tip reflects that in the left side of the heart.
 - ECG.
 - Temperature. A simple non-invasive method of assessing cardiac output and peripheral perfusion is to measure the difference between the peripheral and core temperature. The former is measured by a sensor attached to the big toe, and the latter by a probe placed in either the rectum or the oesophagus. In a warm ambient temperature, the core is higher than peripheral temperature by a gradient that should not exceed 2°C. Any increase in this gradient is a very sensitive indicator of decreased perfusion.
 - Repeated hematocrit and haemoglobin assessment.
 - Blood gases
 - PO₂ is normally between 80-100mm Hg.
 - PCO₂ is normally between 35-45 mm Hg.
4. **Positioning.** Elevating both legs with maintaining the trunk and the remainder of the patient in the supine position is the preferred position in patients with hypovolaemic shock.
5. **Pain relief.**
- If analgesics are needed the intravenous route is used because of the poor absorption from the subcutaneous tissues or the muscles which are hypoperfused.
 - Early immobilization of fractures.
6. **Inotropic agents** are used when the condition fails to improve despite adequate volume replacement and oxygenation. Dopamine and dobutamine are the most widely used. Both improve myocardial contractility while dopamine increases renal blood flow and urine output as well.

Irreversible shock. At some stage, hypovolaemic shock may become refractory to the above therapy. Complete vascular collapse with hypotension unresponsive to volume or drug intervention eventually leads to multiple organ failure (MOF) and lethal central nervous system and cardiac dysfunction.

Irreversibility is difficult to define, but has been related to the duration and volume of haemorrhage, the age and pre-existing cardiovascular fitness of the patient, and the coexistence of massive trauma with multiple direct organ derangement. Before the conclusion that refractory shock has occurred, other causes of failure to respond to therapy should be resolved.

1. Continuing unsuspected blood loss into the chest or abdomen.
2. Inadequate volume replacement.
3. Multisystem trauma with occult thoracic injuries including cardiac tamponade and haemopneumothorax.
4. Acute myocardial insufficiency either from direct injury or secondary to prolonged coronary hypoperfusion.

Cardiogenic Shock

Aetiology

Here there is inadequate blood flow to vital organs due to inadequate cardiac output, despite a normal blood volume. The commonest causes of cardiogenic shock leading to sudden collapse are

1. Acute myocardial infarction (commonest cause).
2. Severe arrhythmias.
3. Massive pulmonary embolism.
4. Cardiac tamponade due to penetrating wounds of the chest.
5. Myocarditis.
6. High spinal anaesthesia, can cause paralysis of the sympathetic supply of the heart.

Clinical features

The clinical manifestations are chiefly caused by the gross reduction of cardiac output. The systolic and diastolic pressures fall, leading to compensatory peripheral vasoconstriction and a cold sweaty skin, inadequate tissue perfusion and increasing metabolic acidosis. Cardiogenic shock is characterized by congested neck veins and a high CVP.

Treatment

1. Oxygen should be administered.
2. Treatment of the cause
 - Myocardial infarction is treated by early thrombolytic therapy and potent analgesics.
 - Control of arrhythmias.
 - Relief of cardiac tamponade by emergency insertion of a needle to drain blood in the pericardium, followed by definitive surgical haemostasis.
3. Mechanical support. The intra-aortic balloon pulsation device serves the following functions
 - Elevating diastolic blood pressure, hence better filling of the coronary arteries.
 - Reduction of myocardial work.

Neurogenic Shock

In neurogenic shock there is paralysis of the vasomotor fibres leading to peripheral pooling of blood and inadequate venous return. It may be due to:

1. Vasovagal attack. This is the simplest type of neurogenic shock. It is due to hearing bad news or watching an unpleasant event. It may also follow severe painful stimuli as a blow to the testis or larynx. Two factors operate to produce the abrupt collapse of the individual. The first is extensive vasodilatation in the splanchnic area causing a sudden reduction in the peripheral resistance, a temporary loss of venous return and a sudden fall in the blood supply to the vital centers. The second factor is excessive vagal stimulation of the heart which causes bradycardia. The mere act of falling to the ground as the individual faints, assists the venous return and helps recovery.
2. Sudden extreme vasodilatation also occurs in patients suffering from a high transection of the spinal cord due to a spine fracture, or as a complication of high spinal anaesthesia or deep general anaesthesia. Shock is caused by loss of sympathetic outflow and consequent vasodilatation. In neurogenic shock there is hypotension, a normal pulse rate or bradycardia and warm dry skin.

Treatment

1. The patient should lie flat, elevation of the legs helps to increase the venous return.
2. I.V. crystalloid solution as Ringers lactate.
3. Vasopressors may be given.

Anaphylactic Shock

This type of shock may follow administration of antibiotics especially penicillina, anaesthetics, sera and dextrans. The antigen unites with the antibodies leading to the release of large amounts of histamine. The patient develops bronchospasm, laryngeal oedema, and respiratory distress. Massive vasodilatation occurs and there is hypotension.

Treatment

1. Intravenous crystalloid infusion.
2. Intravenous hydrocortisona
3. Antihistaminics.
4. Endotracheal intubation may be needed if laryngeal oedema and stridor are developing.

Septic Shock

This is the most lethal type of shock and is recognized as one of the major killers in surgical practice. It is disappointing that despite the availability of more powerful antibiotics, the incidences of septicaemia and septic shock are rising. This is attributed to

- Developing reservoirs of resistant and virulent organisms.
- Concentration of infected patients in critical care units.
- More extensive operations in elderly and poor-risk patients.
- Salvage of severely injured patients.
- Growing population of patients who are immunosuppressed by organ transplantation, and by chemotherapy.

Aetiology

Causative organisms

- Gram-negative bacilli are, by far, the commonest.
- Staphylococci.
- Candida.

Common sources of bacteria

- Peritonitis caused by perforated viscus, gangrenous bowel, or leaking anastomosis.
- Cholangitis or genitourinary infections.
- Infected central venous catheter that may be used for monitoring or for nutrition.

Predisposing factors

All conditions which suppress the immune mechanism predispose to septic shock. These include old age, diabetes mellitus, corticosteroids, chemotherapy, malignancy, and HIV/AIDS.

Pathophysiology

When bacteraemia produces changes in circulation such that tissue perfusion is critically reduced, septic shock develops. This type of shock is the end result of numerous complex interactions between exogenous and endogenous mediators on one side, and the host response to these mediators on the other side.

Mediators cascade

1. The bacterial endotoxin generated by the infecting organisms triggers complex immunologic reactions. This endotoxin is the lipopolysaccharide part of the cell wall of Gram-negative bacilli that is liberated from the dead bacteria.
2. Endotoxin stimulates macrophages and Kupffer cells of the liver to release cytokines. These are large peptides whose normal function is communication to mediate a useful inflammatory response. In huge amounts, however, they have harmful effect on the microcirculation, particularly on the capillary endothelium. Some of these mediators are
 - Tumour necrosis factor (TNF).
 - Interleukines.
 - Platelet activation factor.
 - Prostaglandins (prostacyclin and thromboxane).
 - Nitric acid which is one of the free oxygen radicals and is also a strong vasodilator.

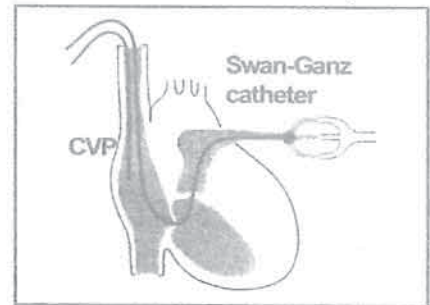


Fig. (6.4) CVP and Swan-Ganz catheters

Microcirculation

1. Septic shock is initially caused by maldistribution of cardiac output. Under the effect of cytokines vasodilatation of the arteries occurs and arterio-venous shunts are opened. The results are
 - a. Reduced peripheral resistance. Nitric acid causes more reduction in the peripheral resistance.
 - b. Capillaries are bypassed and blood is shunted in the oxygenated form from the arteriolar to the venular side.
 - c. Oxygen delivery to the tissues is impaired.
2. Disseminated intravascular coagulation (DIC) is the result of
 - a. Sluggish capillary circulation.
 - b. The action of platelet activation factor.
3. Vascular endothelial damage under the effect of cytokines is the dominant harmful feature of septic shock. The result is leakage of protein-rich fluid from the circulation into the interstitial space causing oedema.

Septic shock
Starts as maldistribution of blood Later myocardial depression & hypovolaemia develops
Common causes Peritonitis Infected venous catheter
Microcirculatory / cellular affection & organ failure are fast to develop
Early signs (hyperdynamic) Fever Flushed warm skin Confusion Hyperventilation Tachycardia
Late signs (hypodynamic) Similar to hypovolaemic shock
Treatment Support resp, circulation, kidneys Fight infection Monitor response
Prognosis is bad

Cellular Derangement

1. Oxygen delivery is reduced because of
 - a. Maldistribution of blood (main factor).
 - b. Late pump failure.
 - c. Hypovolaemia.

When the delivery is reduced below a certain critical level oxygen uptake by the cell is also impaired.
2. Oxygen uptake and utilization by the cells is also directly suppressed by the toxins. The sequence of events that lead from hypoxia to cell death are the same as described under hypovolaemic shock, but they proceed at a faster rate.

SIRS

Septic shock is now considered to be a part of a syndrome called systemic inflammatory response syndrome (SIRS). The cascade of events including mediators release and ending with translocation & bacteria (Fig. 6.5) are common with other members of the syndrome which are

- Major trauma
- Major burns
- Acute pancreatitis
- Neglected hypovolaemia

Acid-base imbalance

As in hypovolaemic shock, there is excess production of lactic acid which produces metabolic acidosis. The body tries to compensate by hyperventilation to wash out CO₂ and by renal conservation of bicarbonates to buffer the acid. These measures temporarily restore pH. Ultimately the kidneys are overwhelmed and fail resulting in the production of frank metabolic acidosis.

The usual changes in arterial blood gases (ABGs) are

- Low pH (lactic acidosis).
- Low PO₂ <70 mm Hg (respiratory distress then failure).
- Low PCO₂ (hyperventilation).
- Low HCO₃ (depleted buffers).

Individual systems and organs

The heart, brain and kidneys are normally protected from swings in blood pressure by autoregulation. In late sepsis, however, these mechanisms fail. In septic shock multiple organ failure (MOF) proceeds faster than in hypovolaemic shock.

1. **Heart.** The heart is privileged by its auto-regulatory mechanisms which maintain coronary blood flow and keeps its ability to extract oxygen in the face of low oxygen delivery level. Initially the heart compensates by increasing its rate and force of contraction, in an attempt to increase the cardiac output and consequently the oxygen delivery to the cells. If septic shock is inadequately treated heart failure takes place because of:
 - Direct effect of some cytokines, e.g. TNF and leukotrienes on the heart.
 - Reduced coronary flow when the blood pressure becomes very low.
 - Increased oxygen demands of the cardiac muscle because of over-work.

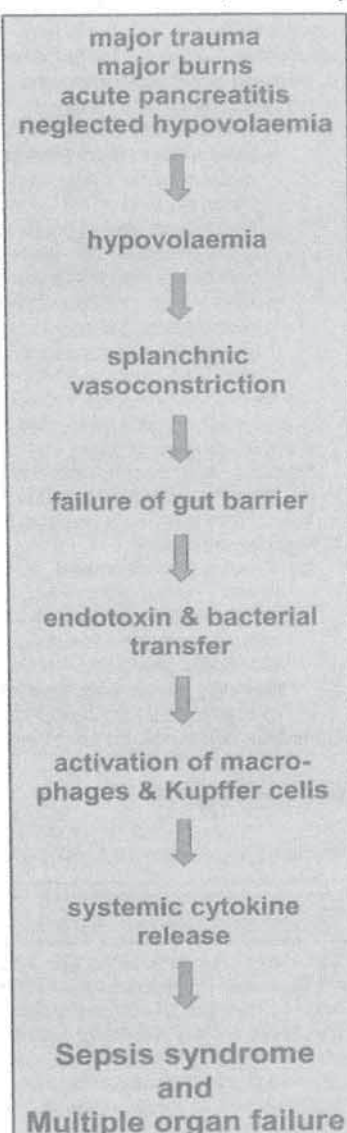


Fig. 6.5. SIRS, sepsis syndrome and multiple organ failure.

2. **Lungs.** Adult respiratory distress syndrome (ARDS) is a common sequel of septic shock. The generalized capillary endothelial damage leads to alveolar and interstitial oedema. The results are:
 - Reduced compliance, and alveoli that are filled with fluid, Both affect ventilation.
 - Opening of arteric-venular shunts that divert blood away from the capillaries impairs perfusion.
 - Interstitial oedema widens the space between blood in the capillaries and air in the alveoli, the so-called thickened alveolo-capillary membrane. This, in addition, impairs gas diffusion.

The three components of respiration, viz., ventilation, perfusion, and diffusion are thus impaired; and ultimately lead to respiratory failure.
3. **Brain.** The brain has also auto-regulatory mechanisms that maintain blood flow if the BP drops mildly. If it drops below 60 mmHg, the compensatory mechanisms are overwhelmed with resultant cerebral ischaemia.
4. **Kidneys.** Changes similar to those described under hypovolaemic shock lead to renal failure.
5. **Liver.** Under the effect of ischaemia and the chemical mediators cholestasis and hyperbilirubinaemia may develop, the so-called "ICU jaundice".
6. **Gastrointestinal tract.** Ischaemia of the gut mucosa produces
 - Gastro-duodenal stress ulceration.
 - Gut barrier failure. The result is translocation of bacteria and endotoxin from the colon lumen to the blood stream and further deterioration of the condition,

Exceptions

- In cardiogenic shock CVP is high. It is the only type of shock where I.V fluids are not given as they will overload the already stressed heart.
- In neurogenic shock pulse is not rapid. The patient may even have bradycardia.

Clinical features

It is useful to describe these manifestations under two stages because diagnosis at the early stage and prompt treatment make a major difference in prognosis.

1. **Hyperdynamic** (warm) septic shock. Diagnosis is difficult and a high index of suspicion is required to detect cases at this early stage. The patient has
 - Restlessness and confusion,
 - Fever above 38°C and chills.
 - Mild reduction in blood pressure.
 - Tachypnoea.
 - Tachycardia.
 - Patient is flushed with warm dry extremities.
 - Oliguria.
 - The cardiac output is elevated.
2. **Hypodynamic** (cold) septic shock. If the previous stage is not treated efficiently, the patient will develop a picture similar to that of hypovolaemic shock with reduced cardiac output.
 - Systolic blood pressure <90 mmHg.
 - Tachycardia and tachypnoea.
 - Cold clammy skin.
 - Oliguria.

Multiple organ failure starts at this stage.

Diagnosis

The diagnosis is helped by

- Polymorphonuclear leucocytosis with abundance of immature forms.
- Finding high lactate level in blood.
- Looking for a source of sepsis.

- Repeated blood culture at the peak of fever or culture from the septic focus will guide antibiotic treatment, but at a later stage. Treatment should be started as soon as possible and should be carried out in an intensive care unit (ICU). Treatment has two main components, viz, support of body systems, and fighting infection. Both should go hand in hand. In addition, monitoring is essential for guidance of treatment.

1. Support of different systems

- a. **Cardiovascular support;** The initial priority in managing septic shock is to maintain a reasonable mean arterial pressure (MAP) to keep the patient alive.
 - Fluid replacement. Prompt correction of fluid deficit is essential. Most of these deficits are properly replaced with a balanced salt solution such as Ringer's Lactate. Any deficiency in red blood cell mass, as evidenced by low hematocrit, can be corrected by transfusion of packed red blood cells. Huge quantities of fluids are often needed to maintain an effective circulating volume. The amount often exceeds 10L within a few hours. The aim is to achieve a CVP of 10-12 cm H₂O, or a pulmonary wedge pressure of 12-15mm Hg.
 - Medications (inotropes and vasopressors). If the patient remains hypotensive despite adequate fluid replacement, as shown by a normal CVP and pulmonary artery wedge pressure, inotropes and vasopressor may be used.
 - Start by norepinephrine or dopamine to keep a mean blood pressure above 65 mm Hg.
 - If no response epinephrine or dobutamine is prescribed.
 - Corticosteroids are helpful if there is poor response to intravenous fluids and vasopressors.
- b. **Respiratory support.** Oxygen administration is essential for all types of shock. Usually 100% oxygen is administered as a start, and is later adjusted according to the response. If the arterial oxygen is mildly reduced oxygen by mask will be sufficient. Reduction of its level below 60 mmHg calls for endotracheal intubation and mechanical ventilation.
- c. **Renal support.** Adequate volume replacement and dopamine administration improve renal blood flow. Haemodialysis is required in case of acute renal failure, until the kidneys recover.

2. Fighting infection

- a. Eradication of sepsis, e.g., drainage of a huge abscess or peritonitis, or resection of gangrenous bowel.
- b. Antibiotics. Aggressive treatment with multiple and broad-spectrum antibiotics is started immediately without waiting for the results of culture and sensitivity. The antibiotic choice is based on the possible suspected organisms which in most cases are Gram-negative bacilli. A combination of cephalosporin, aminoglycoside, and metronidazole can cover the usual organisms. When the results of culture are available, one may change the antibiotic regimen.

3. **Strict control** of blood sugar has been proved to increase the survival.

4. **Prophylaxis** against DVT and stress ulceration is important.

5. Monitoring

Monitoring goes along the same lines as for hypovolaemic shock.

Prognosis

Mortality ranges from 25 to 90%. Death is usually the result of failure to institute therapy soon enough.

Endocrinal Shock

This may occur in patients with Addison's disease or those receiving continuous cortisone therapy if they are subjected to any stressful situation, e.g., infection or surgery. The patient develops severe shock due to failure of release of corticosteroids necessary to cope with the stress from the suppressed adrenal cortex. The result will be a state of peripheral circulatory failure, hyponatraemia and hyperkalaemia. Treatment is essentially prophylactic. Any patient liable to this problem should receive an additional dose of hydrocortisone intravenously prior to any surgical procedure. The treatment of an established case needs large doses of IV hydrocortisone, saline infusions and treatment of the predisposing factor, e.g., infection.

Obstructive shock

May occur in patients with tension pneumothorax, cardiac tamponade or massive pulmonary embolism. It is due to reduced filling of the right side of the heart. The characteristic findings are low cardiac output and high jugular venous pressure and CVP. Unless the specified cause is treated, trials of resuscitation will fail.

COVERED TOPICS AND ANTIBIOTICS

Acute non-specific surgical infections

General principles

Contamination is the mere presence of micro-organisms in a wound. When these organisms invade the tissues and produce ill-effects, this is called infection.

Pathogenesis

The development of any infection depends on interaction between an infectious agent and a susceptible host. In surgical infections an additional factor exists, the presence of a closed unperfused space.

1. **The infectious agent.** Infections in surgical patients can be caused by endogenous or exogenous micro-organisms.
 - Endogenous agents. Many areas in the body possess characteristic microflora. Infection occurs when barriers that separate them from sterile areas of the body are disrupted. Examples include:
 - The skin possesses microflora mainly of Gram positive organisms such as staphylococcus and streptococcus species. These organisms can cause infection of traumatic lacerations or surgical wounds.
 - In the gastrointestinal tract, resident flora of aerobic and anaerobic organisms can be found in the oropharynx, colorectum and distal ileum. This flora can be the source of infections in many surgeries that disrupt the natural barrier between this flora and sterile body areas.
 - Exogenous agents. Many microbes within the external environment can become pathogens. Most important is the exogenous source that lies within the hospital and is responsible for hospital acquired infections. Sources include other patients, staff members and faults in sterilization systems.
2. **Host reaction.** The host's immune mechanisms include
 - Non-specific immunity. This function is carried out by phagocytic leucocytes which migrate from the blood stream towards the site of organisms by a process of chemotaxis in which complement plays a role. They adhere to the organisms and engulf them in a phagocytic vacuole. Corticosteroids and malnutrition reduce the number of granulocytes that arrive at a contaminated

CHAPTER CONTENTS

- Acute non-specific surgical infections — General principles
- Individual acute non-specific surgical infections
 - Surgical site infection
 - Cellulitis
 - Erysipelas
 - Boll (furuncle)
 - Carbuncle
 - Hydradenitis suppurativa
 - Acute abscess
 - Acute lymphangitis and lymphadenitis
 - Bacteraemia and septicaemia
- Acute specific surgical infections
 - Tetanus
 - Gas gangrene
 - Necrotizing fasciitis
- Chronic specific infections
 - Tuberculosis
 - Syphilis
- Parasitic infections
 - Schistosomiasis
 - Filariasis
 - Amoebiasis
 - Echiniceccosis (hydatid disease)
- Fungal infections
 - Candidiasis
- Antibiotics in surgical infections
- Decontamination

- Staphylococci produce coagulase enzyme, which limits infection, and tend to produce pus. Streptococci tend to produce spreading infection.
- Antibiotics are useful when infection is spreading through the tissues, e.g., cellulites and erysipelas.
- Drainage is essential when abscess formation has occurred.
- Suspect diabetes in cases with recurrent infections.

site and, thus, reduce immunity. The next step is killing the phagocytosed organisms either by lysosomal enzymes or by oxidative killing. It was found that leucocytes in uncontrolled diabetics migrate, adhere and kill bacteria poorly and that these functions are improved with control of diabetes.

- Specific immunity. This is carried out by specific antibodies produced on prior exposure to antigen with subsequent activation of T and B lymphocytes. Defects in specific immunity, as seen in patients on immunosuppressive therapy, acquired immunodeficiency syndrome (AIDS) and agammaglobulinaemia, result in increased susceptibility to surgical infections.
3. **The closed space.** Most surgical infections start in a susceptible poorly vascularized area such as a wound or a natural space. Poor perfusion, hypoxia, hypercapnia and acidosis all predispose to infection. Natural spaces with narrow outlets such as the gall bladder, appendix and diverticula are especially prone to become obstructed and then infected.

Bacteriology

1. **Staphylococci** These are the commonest organisms seen clinically. They are Gram-positive cocci that live in the skin including the sebaceous and sweat glands, and in the nostrils in about 25-50% of normal adults. Staph. aureus infection gives rise to pustules, boils, carbuncles, whitlows and paronychia. It is the commonest cause of osteomyelitis and breast abscess. It is commonly seen in infected surgical wounds and is responsible for a serious type of pneumonia. Staphylococci produce a number of toxins, eg., coagulase, leucocidin, and alpha trypsin. Some strains also produce the enzyme penicillinase, which renders them resistant to penicillins. Some of the most resistant bacteria are the so called methicillin resistant Staph aureus (**MRSA**) which pose a big problem, particularly in hospital-acquired infections. Most of such strains are sensitive to vancomycin, others are vancomycin resistant (**VRSA**).
2. **Streptococci** These are Gram-positive organisms that grow in chains. For practical purposes, two types of streptococci need to be considered, the haemolytic and non-haemolytic. Haemolytic streptococci are often present in the nasopharynx and are readily transmissible by droplet infection from person to person. They can cause spreading infection such as cellulitis, lymphangitis and erysipelas. The non-haemolytic streptococci have much lower virulence. They are normally found in the mouth and the bowel (enterococcus). They are commonly responsible for low grade infective processes such as dental sepsis.
3. **Aerobic Gram-negative bacilli**
 - *Escherichia coli* (*E. coli*). They are found in the intestinal tract of all human beings. Many varieties are non pathogenic under ordinary conditions. *E. coli* are responsible, either alone or in mixed infection, for the majority of suppurative lesions within the abdomen. *E. coli* is also responsible for most urinary tract infections.
 - *Klebsiella*. This organism represents a special variety of encapsulated gram-negative bacilli. They are found in the respiratory tract and can spread from case to case and cause fatal pneumonia in debilitated patients.
 - *Pseudomonas aeruginosa* (*pyocyanea*) is present in human faeces in 20 percent of cases. It can act as a primary cause of infective processes or much more commonly as a secondary invader especially in open wounds and burns. It is recognized by the blue-green colour of the pus and its odour. Once introduced into a ward, *pseudomonas* is very difficult to eradicate.

- **Proteus.** *Proteus vulgaris* is a common cause of urinary tract infection and can also occur in wounds and burns as a component of mixed infection.
- 4. **Anaerobic bacteria** Obligatory anaerobes, chiefly peptostreptococci and gram-negative bacilli (bacteroids), are found in the normal flora of the skin and mucous membranes. When the epithelial barrier is disturbed, these organisms particularly bacteroids, can invade tissues and produce abscesses or enter the blood stream and cause septicaemia. These abscesses are characterized by gas formation, tissue necrosis and foul smelling discharge. Aerobic cultures fail to grow these organisms. Postoperative anaerobic wound infection usually follows operations on the colon, oral cavity or the vagina.

Complications

1. Spread of infection occurs by several mechanisms

- a. **Direct**
 - **Necrotizing infections.** These tend to spread along anatomical paths. Necrotizing fasciitis, for example, spreads along poorly perfused fascial and subcutaneous planes while its toxins cause thrombosis of large vessels ahead of the necrotic area resulting in more ischaemic vulnerable tissue.
 - **Abscesses** If they are not properly drained, abscesses enlarge and destroy surrounding tissues.
 - **Phlegmons.** These contain little pus but much edema. They spread along fat planes and by contiguous necrosis.
 - b. **Lymphatic spread.** Streptococcal and occasionally staphylococcal infections spread along lymphatic vessels producing lymphangitis with characteristic red streaks in the skin that travel proximally.
 - c. **Blood stream spread.** This may result in distant abscesses such as liver abscesses in-patients suffering from appendicitis or inflammatory bowel disease. These are called pyaemic abscesses and they are due to septic thrombophlebitis of the draining veins. Bacteraemia or septicaemia may also result.
2. **Fistulae and sinus.** A neglected anal abscess, for example, ends in an anal fistula while a pilonidal abscess ends in mere sinus tract.
 3. **Necrosis or gangrene** of the affected part.
 4. **Suppressed wound healing.** Continued infection results in stimulation of collagenase production by bacteria. This suppresses wound healing.
 5. **Immunosuppression and superinfection** Toxicity from uncontrolled infection may result in immunosuppression and this in turn will facilitate superinfection with resistant organisms.
 6. **The systemic inflammatory response syndrome (SIRS) and multiple organ failure.** This is the most serious complication of acute surgical infections and is caused by major sepsis. It usually results from release of endotoxins by Gram-negative organisms leading to septicaemia. This has been discussed in chapter 6. The prognosis of septicaemia is related to the age and general health of the patient and to the number of affected organs. If one organ is affected (usually the lung or kidney), 70% of patients recover. If two organs are involved, the recovery rate drops to 50% while if three organs or more are affected the prognosis is worse.

Diagnosis

1. **Clinical examination** This is the easiest way to establish the diagnosis.
 - Locally the inflamed area is painful, red, hot and tender with loss of function.
 - The draining lymph nodes may be enlarged, painful and tender.
 - Constitutional symptoms include fever, headache, malaise, furred tongue and tachycardia. Dyspnoea and rigors may occur in severe cases indicating septicaemia or pyaemia.

2. Laboratory studies

- General findings. There is mild to moderate leucocytosis with prevalence of immature granulocytes (shift to the left). In overwhelming sepsis there may be leucopenia and possibly findings related to disseminated intravascular coagulation together with impaired hepatic, renal and respiratory functions.
- Bacteriological studies. Bacteriological examination of discharge, and culture and sensitivity tests play an important role in diagnosis. The patient should not be on antibiotics for 3 days before the sample is taken. Blood cultures are essential in serious infections. Usually three blood samples are taken over 24 hours period and the blood is used for aerobic and anaerobic cultures.

3. Imaging studies

- Plain radiographs are helpful in the diagnosis of several types of surgical infections including pulmonary infection, subphrenic infection (elevated immobile cupola of the diaphragm), psoas abscess (obliteration of the psoas shadow), and osteomyelitis.
- Other imaging studies, such as ultrasonography, CT scanning and radionuclide scans are very helpful in localizing deep seated infections.

Principles of treatment

1. Incision and drainage. Once pus is formed, it should be drained.
2. Excision. Some surgical infections may be excised, e.g., an infected appendix. Gas gangrene infections may require amputation of the affected limb. Debridement of dead tissues in any wound is important.
3. Antibiotics. Simple surgical infections that respond to incision and drainage alone, such as furuncles and acute abscesses, do not usually require antibiotics after drainage. Infections likely to spread or persist require antibiotics, preferably on the basis of sensitivity tests.
4. General supportive measures. Predisposing factors should be corrected to help the body combat infection. These include such measures as control of diabetes, limb revascularization in ischaemia, and nutritional support.

Individual acute non-specific surgical infections

Surgical site infections (SSIs)

SSIs are infections of the tissues, organs or spaces exposed during the performance of an invasive surgical procedure. SSIs are classified into

- **Incisional** there are either superficial limited to the skin and subcutaneous tissues or deep involving the musculoaponeurotic layers.
- **Organs.**
- **Spaces** e.g. subphrenic, pelvic or interloop abscesses (Chapter 36).

The development of SSIs is due either to general factors related to the patient or local factors related to the wound.

General host factors

- Older age.
- Obesity.
- Anaemia and malnutrition.
- Immunosuppression as in diabetes mellitus, uraemia and malignancy.
- Intake of drugs which suppress the immune system as corticosteroids, chemotherapy and immunosuppressive drugs.

Local factors

- Poor skin preparation.
- Defect in sterilization of the instruments or lack of adherence to theatre discipline.
- Prolonged surgical procedures.
- Improper surgical technique e.g. leaving dead spaces, haematoma, too much use of diathermy or poor handling of tissues.
- Poor blood supply prolonged periods of hypotension and suture of tissues under tension predispose infection.
- Nature the operation this is the most important factor which determines SSIs as most of these infections are endogenous due to microorganisms from the patient himself. Surgical wounds are classified based on the presumed magnitude of the bacterial load at the time of surgery into:
 - o **Clean wounds (Class I):** These are elective non traumatic wounds in which the gastrointestinal, urinary or respiratory tracts are not entered.
Risk of SSI → less than 2%.
 - o **Clean contaminated (Class II):** Elective surgery into the gastrointestinal urinary or respiratory tracts with no significant spillage.
Risk of infection → 2-5%.
 - o **Contaminated wounds (Class III)**
 - Open accidental wounds encountered within 4 hours.
 - Gross spillage from the gastrointestinal tract.
 - Incision through non-inflamed, non-purulent tissues
Risk of infection → 10-20%
 - o **Dirty wounds (Class IV)**
 - Traumatic wounds more than 4 hours.
 - Purulent infection or necrotizing soft tissue infection.
 - Perforated viscus accompanied by high degree of contamination.
Risk of infection → up to 40%.
- Presence of foreign bodies or prosthetic material.

Clinical features

- Surgical site infection usually appears between the fifth and tenth days postoperatively but may sometimes appear earlier or later.
- Wound pain and postoperative fever are the earliest manifestations.
- The wound is swollen (sutures are dipping through the skin), tender and red.
- Fluctuant areas or crepitus can occasionally be felt.
- Discharge from the wound may be seen.
- Infections deep to the deep fascia may be difficult to recognize. They are associated with systemic signs of infection.

Recommendations to minimize surgical site infections

The patient

1. Correct any predisposing factors as control of diabetes, stopping of smoking and correction of nutritional deficiency.
2. Operations are avoided in patients with active infections if possible.
3. Shaving or clipping the hairs just before skin incision.
4. Skin preparation with appropriate antiseptics.

The surgeon

1. Surgeon should have short nails and should scrub properly.
2. Meticulous surgical technique by proper haemostasis, gentle handling of tissue and avoiding tight sutures or leaving dead space.
3. Delayed primary closure of heavily contaminated wounds.

Prophylactic antibiotics

Evidence based surgical practice has shown that for contaminated and clean-contaminated wounds, postoperative infection can be avoided by using appropriate prophylactic antibiotics.

The choice of prophylactic antibiotics

- Select the antibiotics active against the organism likely to be encountered in the hospital.
- Select the antibiotics which give high concentrations in the required tissues used for a suitable time.
- Select the antibiotics which are unlikely to induce renal or hepatic damage. If such damage already occurs, correct dosing regimens should be used.

The evidence that newer expensive broad spectrum antibiotics are more effective for prophylaxis than the cheaper narrower spectrum antibiotics is weak.

There are many protocols recommend; the following is a scheme advised for prescription of prophylactic antibiotics.

Clean operations No need for prophylactic antibiotics except in

- When an implant, e.g. prolene mesh or a vascular graft is used.
- In patients with valvular heart disease to prevent infective endocarditis.
- In emergency surgery in a patient with preexisting active infection.
- When infection would be very severe or have life-threatening consequences as in aortic surgery or organ transplantation.

Recommendation

- One dose of 1st generation cephalosporin, or (ampicillin + sulbactam)

Clean contaminated operations

- One dose of second generation cephalosporine or (ampicillin + sulbactam) + Aminoglycoside.

Contaminated operations as contaminated, but add Metronidazole.

In all if the operation exceeds 2 hours, another dose is prescribed.

Treatment

- Liberal drainage. The wound should be opened by removal of skin stitches.
- Antibiotics are used in invasive infections guided by culture and sensitivity tests.
- Possible sources of hospital acquired infection should be traced and corrected.

Cellulitis

Aetiology

Cellulitis is often caused by the introduction of Gram-positive bacteria mostly streptococci or occasionally staphylococci into the superficial skin structures. The portal of entry may be trivial, e.g. scratch or prick.

Pathology

Cellulitis is an invasive non suppurative infection of the loose connective tissue. Although polymorphonuclear leucocytes are abundant, there is no pus formation.



Fig. 7.1. Cellulitis of the face.

Clinical features

- The affected area is red (Fig. 7.1) or reddish brown, indurated, hot and painful.
- It spreads rapidly and the advancing edge is ill defined.
- The skin may be the seat of blisters.
- A moderate or high fever is always present.
- Lymphatic spread produces red streaks of lymphangitis and enlarged tender regional lymph nodes.
- There is no suppuration (no pus formation).

Differential diagnosis

- Contact allergy.
- Chemical inflammation at the site of drug injection.
- Deep vein thrombosis.

Treatment

- Antibiotics (penicillin group).
- Rest and elevation of the affected part.
- Hot packs.

If no response has occurred after 48 hours either the diagnosis of cellulitis is doubted and an abscess is suspected, or resistant organisms as staphylococci are involved.

Erysipelas

This is a rapidly spreading non-suppurative inflammation of the lymphatics of the skin caused by a specific strain of haemolytic streptococci. The organisms gain access through a minute scratch or abrasion.

Clinical features

- Symptoms of toxæmia are present.
- Locally the picture is similar to cellulitis, but there are some differences
 - The skin is rose-pink.
 - The edge is well-defined, slightly raised and often shows minute vesicles just beyond the spreading margin.
 - There may be islets of inflammation beyond the spreading margin separated from the main area by apparently normal skin.

Complications

- Facial erysipelas may lead to cavernous sinus thrombosis.
- Septicaemia,
- Recurrent erysipelas may block the lymphatics leading to elephantiasis.

Treatment

The patient is isolated because the disease is very contagious. Otherwise, treatment is similar to cellulitis.

Boil (furuncle)

Aetiology

- This is a staphylococcal infection of a hair follicle or a sebaceous gland.
- It can occur in any hirsute area of the skin and is particularly common in the face, neck (Fig. 7.2) and axilla.
- Boils are more common in diabetics and whenever there is lack of personal hygiene.

Clinical features

Clinically a boil forms a small painful indurated swelling which is hot, red and very tender. Early treatment may allow the boil to resolve without suppuration (Blind boil). More often, necrosis of the central part occurs and is discharged together with a small bead of pus.

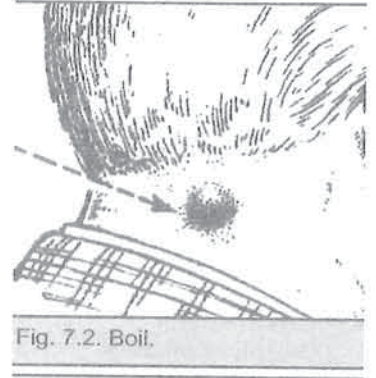


Fig. 7.2. Boil.

Treatment

- Antibiotics effective against staphylococci.
- Icthiol ointment and warm fomentations facilitate necrosis of the overlying skin and escape of pus and slough.
- Painting the surrounding skin with an antiseptic to prevent infection of neighbouring glands or hair follicles.
- Always suspect diabetes mellitus in patients who develop recurrent boils.



Fig. 7.3. Multiple sinuses of a carbuncle and incision to remove sloughs.

Carbuncle

This is infective gangrene of the subcutaneous tissues usually secondary to infection by *Staphylococcus aureus*. It is common in immunocompromised patients as diabetics. The infection occurs mainly in the face, nape of the neck and the back.

Pathology

Infection usually starts in a hair follicle and extends to the subcutaneous fat where other hair follicles get the infection. Multiple areas of necrosis and thrombosis of blood vessels occur. Patches of skin undergo sloughing and separate from the underlying granulation tissue.

Clinical features

There is usually severe toxæmia. The carbuncle starts as a painful induration of the skin and subcutaneous tissues. The skin is red. As the swelling enlarges, its central part becomes soft but usually no fluctuation can be elicited. Multiple areas of skin thin out and separate forming multiple sinuses (Fig. 7.3) through which multiple sloughs separate slowly. Usually no frank pus forms.

Complications

1. Local spread of infection leads to cellulitis and lymphangitis.
2. Pyæmia and septicaemia.

3. Carbuncles of the dangerous areas of the face can lead to cavernous sinus thrombosis.

Treatment

1. Antibiotics effective against resistant staphylococci.
2. Culture and sensitivity of the discharge.
3. Proper control of diabetes mellitus, if present.
4. Glycerine magnesias local fomentations help separation of the sloughs.
5. Excision of sloughs (Fig. 7.3).

Hydradenitis suppurativa

Mixed Staph. and streptococcal infection of the apocrine sweat glands, in the perineum or the axilla, produces multiple abscesses and pus-discharging sinuses. Infection is difficult to eradicate and the case usually progresses to chronicity with considerable fibrous tissue reaction.

In the perineum, hydradenitis suppurativa is often misdiagnosed as multiple anal fistulae. Careful examination reveals the absence of internal anal openings. Treatment by drainage of abscesses followed by careful hygiene, painting with disinfectants and antifungal applications may be enough. Otherwise, excision of the apocrine sweat-bearing skin followed by skin grafting is needed.

Acute abscess

An abscess is a localized suppurative inflammation. It is caused by pyogenic organisms. The commonest causative organisms are staphylococci that produce a coagulase enzyme which helps localize the acute inflammatory processes.

Pathogenesis

The organisms reach the tissues by

1. Direct access through wounds, scratches, and abrasions, or along natural passages such as lactiferous ducts.
2. Local extension from an adjacent focus, e.g. osteomyelitis of the jaw from an infected tooth.
3. Lymphatic spread. Infection reaches the regional lymph nodes along lymphatics from a septic focus in their drainage area.
4. Blood spread. Organisms gaining access into the circulation, as in bacteraemia or pyaemia, may lodge in distant tissues and cause abscesses e.g. pyaemic liver, and lung abscesses.

Pathology

Grossly. An abscess consists of three zones

1. A central zone of coagulative necrosis. This ultimately separates from surrounding tissues and forms a slough which becomes liquified by the enzymes of dead leucocytes. An abscess cavity forms containing plasma cells and inflammatory exudate. The resulting opaque fluid is called pus and is composed of inflammatory exudate, dead leucocytes, necrotic tissue and dead and living organisms.
2. An intermediate zone of granulation tissue forms a protective layer against the spread of bacteria and their toxins.
3. A peripheral zone of acute inflammation fades gradually into healthy surrounding tissues.

Fate

1. Resolution occurs if resistance is high and treatment is started early before pus forms.
2. Pointing and rupture is the commonest sequel. The abscess discharges its pus along the plane of least resistance.
3. Spread of infection either locally, by lymphatics, or by blood stream.
4. Chronicity. A dense fibrous tissue reaction forms around the incompletely resolved abscess leading to the formation of a mass with little inflammatory reaction around. This occurs when an abscess is treated by a prolonged course of antibiotics or if improperly drained.

Clinical features

An abscess starts as a painful tender mass. The covering skin is red, and oedematous. The draining lymph nodes are usually enlarged and tender. There is usually a systemic reaction in the form of fever, malaise, headache, tachycardia and anorexia. When pus forms due to liquefaction of the necrotic center, the following changes are noted

1. The pain becomes throbbing.
2. The fever becomes hectic.
3. The covering skin shows pitting oedema.
4. The inflammatory reaction becomes localized.
5. Fluctuation can usually be elicited in a subcutaneous abscess. This sign is absent or difficult to elicit in deep abscesses (e.g. perinephric or gluteal abscess), in abscesses covered by strong fascia (e.g. parotid abscess) and in breast abscesses as the normal breast tissue gives a false sense of fluctuation.
6. There is shooting leucocytosis with shift to the left.

Treatment

Once pus forms, the only treatment should be free drainage.

1. Incision and drainage is the standard method. The incision should be situated over the pointing part of the abscess and should be dependent, otherwise a counter incision over a dependent area is necessary. The loculi inside the abscess are broken to ensure adequate free drainage and a specimen of the pus is sent for culture and sensitivity. A rubber drain or a wick of gauze is left protruding from the abscess cavity. The drain serves two purposes; it allows escape of discharge, and prevents premature closure of the skin wound before the abscess cavity is obliterated by granulation tissue. Adequate free drainage is usually enough for most abscesses and antibiotics are required only in overwhelming infections or in immunocompromised patients. In dangerous areas, e.g. the neck, a sinus forceps is used to open the abscess after the skin incision (Hilton's method, Fig. 7.4)). This avoids injury of important structures in the area.
2. Ultrasound or CT-guided aspiration drainage is applicable for deep seated collections, e.g., an intraperitoneal abscess.



Fig. 7.4. Hilton's method of abscess drainage.

Acute lymphangitis and lymphadenitis

- Acute lymphangitis is due to infection of lymph vessels by organisms, usually streptococci, entering through an abrasion or a wound in the area drained by lymphatics. If the main trunks are affected they appear as red streaks in the overlying skin (tubular lymphangitis). There is usually fever and rigors.
- Acute lymphadenitis is due to spread of infection along lymphatics from a septic focus in the drainage area. The affected lymph nodes are painful, enlarged and tender. Fever,

malaise and signs of toxaemia are usually present and the primary focus of infection may be detected in the drainage area.

Treatment

Antibiotics and hot applications are used in both lymphangitis and lymphadenitis. If suppuration occurs in the lymph nodes, incision and drainage are essential.

Bacteraemia and septicaemia

- **Bacteraemia** denotes the asymptomatic presence of bacteria, which are not multiplying in the blood. Its significance is variable but it is usually harmless. It usually follows dental work and instrumentation of the urinary tract especially in the presence of infection. Bacteraemia is hazardous in patients with damaged heart valves or with prosthetic valves because micro-organisms may settle on these valves or prostheses causing severe damage. Bacteraemia is also dangerous in patients with immuno-suppression. Antibiotic prophylaxis is essential in such cases. It should be noted that Gram-positive bacteraemia is usually less significant than Gram-negative bacteraemia.
- **Septicaemia** means the presence of multiplying organisms in the blood stream plus leucocytosis. Sepsis is the clinical reflection of bacterial infection. It usually denotes significant infection in which bacteria, bacterial toxins or inflammatory mediators escape the control of the immune system, enter the blood stream and produce a systemic response including chills, fever and sometimes pulmonary failure or shock.

Acute specific surgical infections

Tetanus

Tetanus is a specific anaerobic infection that is mediated by the neurotoxin of *Clostridium tetani* and leads to nervous irritability and tetanic muscular contractions. The disease is becoming less prevalent thanks to the compulsory immunization in childhood.

Aetiology

Organism. *Clostridium tetani* is a gram positive anaerobic bacillus with a terminal spore giving the characteristic drum-stick appearance. The organism are naturally present in the intestine of horses and the spores are present in manured soil and street dirt,

Mode of infection

1. **Wounds.** The causative organism enters and flourishes in hypoxic wounds contaminated with soil or faeces. The tetanus-prone wound is usually a puncture wound or one containing devitalized tissue, a foreign body, or associated pyogenic organisms.
2. **Umbilical stump.** Tetanus neonatorum arises from infection of the umbilical stump by contaminated dressings or powders

Pathology

The bacillus remains at the site of inoculation but its exotoxin reaches the central nervous system along the blood stream, the motor nerves or both. Once the toxin reaches the nervous system, it is at once fixed by the motor cells and can not be detected in the blood or CSF Tetanus antitoxin can only neutralize the toxin before it gets fixed to the nervous tissue.

The toxin increases the excitability of the motor cells of the medulla and spinal cord, so the slightest stimuli produce violent spasm. Death results from exhaustion, hyperpyrexia, heart failure, asphyxia or pneumonia.

Clinical features (Fig. 7.5)

Incubation period. The incubation period is variable and depends on previous immunization. In the non-immunized the period is short and varies from 24 hours to 15 days. In immunized patients the period is longer than 11 days and may be several weeks or months.

Tonic stage. The first symptoms are usually pain and tingling in the area of injury, limitation of movements of the jaw (lock jaw, trismus) and spasm of the facial muscles (risus sardonicus). These are followed by stiffness of the neck, difficulty in swallowing, and laryngospasm. Hesitancy in micturition due to sphincter spasm is also seen.

Clonic stage. In more severe cases reflex paroxysms of violent muscular contractions become superimposed on the above mentioned tonic rigidity. The spasms are initiated by any form of stimulation such as bright light and are always tonic as well as clonic so that relaxation is incomplete during the intervals between clonic contractions. During these spasms the body may be arched backwards (opisthotonos) forwards and laterally. Spasms become more frequent and involve more and more muscle groups. As spasms of the intercostal muscles and diaphragm occur, longer and longer periods of apnoea follow. The temperature is normal or slightly elevated. Sweating is profuse. Marked tachycardia is a grave sign. Attacks vary in severity and some are very mild.

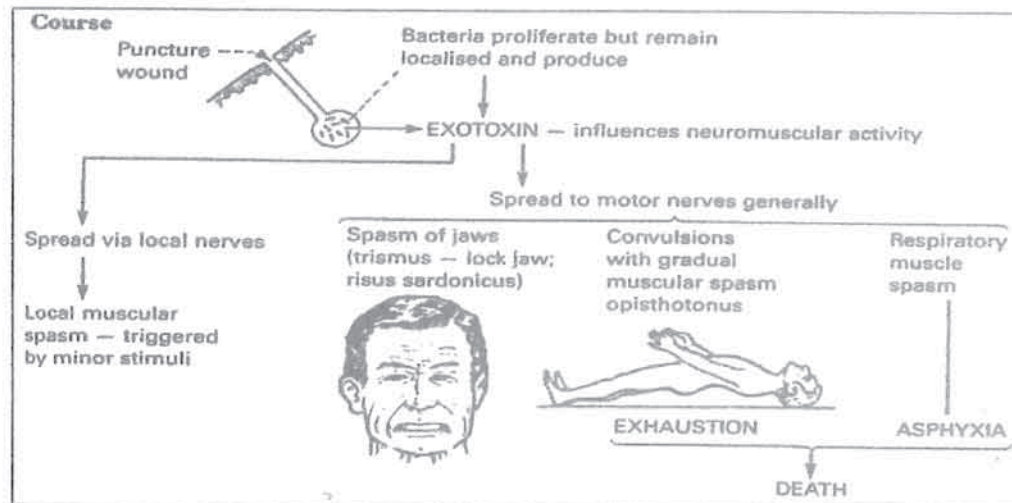


Fig. (7.5) Clinical features of tetanus

Laboratory findings

Polymorphnuclear leucocytosis may be present.

Differential diagnosis

1. Trismus due to local causes such as impacted wisdom tooth or temporomandibular arthritis. This can be excluded by careful local examination,
2. Meningitis. The neck muscles are affected first and the CSF is turbid and contains leucocytes and organisms.
3. Strychnine poisoning: The spasms start in the extremities and are entirely clonic with complete relaxation during the intervals.
4. Tetany. The hands and feet are mainly affected, Chvostek's sign is positive and, serum calcium is low.
5. Rabies: There is history of a dog-bite. The spasms occur on seeing and drinking water. The muscles of deglutition and respiration are mainly affected.

Prevention

- Every child should be actively immunized with tetanus toxoid, beginning with routine childhood immunization and continuing with booster injections every 7-10 years.
- Individuals who previously received three or more doses, the last within 10 years, need only a booster dose of tetanus toxoid on exposure to tetanus prone wounds (0.5 ml IM).
- Individuals not previously immunized should receive the following doses
 1. Clean minor wounds (tetanus is unlikely) 0.5 ml of tetanus toxoid is given as the initial dose. Two further injection are given at 4 weeks intervals.
 2. Wounds with high tetanus risk. The initial immunizing dose (0.5 ml intramuscularly) of the tetanus toxoid is given together with 250 units intramuscularly of tetanus immune globulin (TIG) in a different syringe and at a different site, and consider the use of antibiotics. Plan to complete the toxoid series. Equine antitoxin should be used only if TIG is not available and only after testing for hypersensitivity to the product.

Treatment

Intensive treatment should be started soon, as respiratory paralysis may advance rapidly.

1. Neutralize toxin with TIG. The dose in established cases is 3000-6000 units intramuscularly, given preferably in the proximal portion of the wounded extremity or in the vicinity of the wound. Repeated doses may be needed since the half-life of the antibody is about 3 weeks and established tetanus often lasts longer.
2. Excise and debride the suspected wound under anaesthesia. Surgery should be done after systemic serotherapy has been started. The wound must be left open and may be treated with hydrogen peroxide.
3. The patient should be protected from sudden stimuli, unnecessary movements and excitement. Barbiturates are used cautiously as they often cause cardiorespiratory failure. Diazepam may reduce the dose of barbiturates needed to control spasms. Curarization with mechanical ventilation is a better alternative to large cardio-suppressant doses of barbiturates.
4. The patient with respiratory problems may require tracheostomy, since mechanical ventilation, once it becomes necessary, must be continued for weeks. The patient should be intubated once respiratory problems appear.
5. Aqueous penicillin G, 10-40 million units a day, by intermittent intravenous bolus injection should be given to kill clostridial organisms and prevent further release of toxin.
6. Nursing. The patient is isolated in a dark quiet room, and nutrition is maintained by a nasogastric tube. All doses should be modified in tetanus neonatorum.

Prognosis

The death rate is 30-60% in established tetanus with respiratory insufficiency. The death rate is inversely proportionate to the length of the incubation period and directly proportionate to the severity of symptoms. One attack of tetanus does not confer a life-long immunity. Patients who recover from an attack need to have an active immunization schedule.

Gas gangrene (Clostridial myositis)

Aetiology

Gas gangrene is an acute spreading gangrene, associated with gas formation and profound toxæmia, due to infection of extensive wounds by the anaerobic spore-bearing bacillus *Clostridium perfringens* (welchii) and other *Clostridia* species. Gas gangrene is closely associated with grossly contaminated war injuries.

Factors predisposing to gas gangrene

1. Lacerated wounds involving bulky muscles as in the gluteal area and thigh (Fig. 7.6).
2. Presence of foreign bodies or devitalized tissues.
3. Ischaemia of the muscles, e.g. due to injury of the main vessels, tight bandages or casts or sutures under tension.
4. Infection by aerobic bacteria which make the medium suitable for the anaerobic clostridia.
5. Gas gangrene may follow above knee amputation (Fig. 7.7) especially in elderly persons who may suffer from faecal incontinence leading to infection of the stump.

Pathology

Clostridia proliferate and produce toxins that diffuse into the surrounding tissue. The toxins destroy local microcirculation. This allows further invasion, which can advance at an astonishing rate. The alpha toxin, a necrotizing lecithinase, is particularly important in this sequence, but other toxins including collagenase, hyaluronidase, leukocidin, protease, lipase and haemolysin also contribute.

Clostridial toxins induce

1. Muscle necrosis, fermentation and gas production.
2. Haemolysis and mild jaundice.
3. Degenerative changes in the liver, kidneys and adrenals.

Clinical features

Incubation period varies from few hours to few days.

Generally

- The patient is pale, anxious and apprehensive.
- The temperature may be raised and there is marked tachycardia.
- The hands are cold and clammy.
- An icteric tinge may be present and there is oliguria.
- In severe cases the patient is shocked.

Locally

- The patient complains of pain and numbness in the affected area.
- The wound is swollen and there may be crepitus with gas bubbles (Fig. 7.7).
- A sanguinous discharge of a characteristic odour may exude from the wound.
- The affected muscles show brick red then greenish and finally black discolouration. They do not contract on pinching and do not bleed if cut.
- The skin overlying the wound may show greenish or black discolouration and there may be multiple blebs full of a foul smelling, dark fluid.

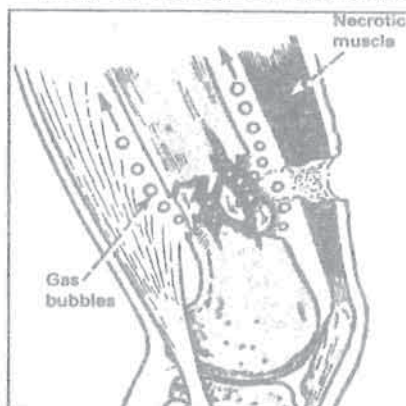


Fig. (7.6) Gas gangrene

The proper diagnosis of clostridial gas gangrene depends mainly on the clinical appearance of the wound and the presence of large Gram-positive rods on stained smears of exudate or tissue.

Clostridial mycrosis (gas gangrene) is preventable, mainly by proper debridement of deep lacerated wounds.



Fig. (7.7) Gas gangrene in amputation stump

Differential diagnosis

1. Non-Clostridial gas-producing infections are usually caused by a mixture of Gram-negative bacilli (e.g., *E. coli*) and Gram-positive cocci. These infections are not as virulent as gas gangrene and respond well to incision and drainage.
2. Surgical emphysema is due to the presence of gas under the skin. It is not associated with signs of toxemia or peripheral circulatory failure and the wound looks healthy,

Prevention

All clostridial infections are preventable.

1. Wound debridement. Wounds occurring outdoors and contaminated with soil, foreign bodies, faeces or dirt or associated with extensive muscle injury should be examined under anaesthesia. All dead muscles (do not bleed on cutting and do not contract on pinching) should be excised and the wound is cleaned, and left open.
2. Antibiotics especially penicillin are valuable but are in no way a substitute for proper surgical debridement.
3. Circulatory support in severe injuries to avoid tissue hypoxia that renders the patient more susceptible to clostridial infections.

Treatment

1. Wound management. This is the most important step. Under general anaesthesia, the wound is opened and all dead tissues are excised, Tight fascial compartments are decompressed. Dead muscles are debrided adequately. The deep fascia and skin are left open. When there is diffuse myositis with complete loss of blood supply or when adequate debridement leaves a useless limb, amputation becomes necessary.
2. When the source of the clostridial infection is an associated injury of the colon or rectum, a proximal diverting colostomy is essential together with local management. Diverting colostomy is also indicated in extensive perineal infections to avoid gas gangrene.
3. Hyperbaric oxygenation. Hyperbaric oxygen inhibits bacterial invasion and the production of alpha toxin. Hyperbaric oxygen at 3 atm is given for 1-2 hours and is repeated every 6-12 hours. Three to five exposures are usually necessary.
4. Antibiotics. Penicillin, 20-40 million units/day, is given intravenously. In patients allergic to penicillin, clindamycin or metronidazole can be used. In mixed infections one can use multiple antibiotic combinations, Anti gas gangrene serum is no more used in modern surgical practice.

Prognosis

- Mortality. Death rate is currently about 20%. Mortality is higher with delay of treatment, associated severe injuries, advanced disease with invasion of vital structures and pre-existing illness.
- Limb salvage (saving). With established clostridial myositis, the rate of salvage of functioning limbs is not favourable, the myonecrosis added to the original injury render it essential in many cases to perform a life-saving amputation.

Necrotizing fasciitis**Aetiology**

This is an invasive infection that is usually caused by a mixed microbial flora including microaerophilic streptococci, staphylococci, Gram-negative bacteria and anaerobes, especially peptostreptococci, and bacteroids. The organisms usually gain

access through a puncture wound, leg ulcer or a surgical wound. This infection is more likely to occur in immunocompromised patients, e.g., diabetics and patients with malignant neoplasms.

Pathology

The infectious process spreads along the fascial planes and results in infectious thrombosis of the vessels passing between the skin and the deep circulation. Superficial skin necrosis follows. Haemorrhagic bullae appear as the first sign of skin death. Fascial and subcutaneous fat necrosis involves an area wider than the skin.

Clinical features

The patient is alert and fully conscious. There are manifestations of toxæmia with fever and tachycardia. The skin shows haemorrhagic bullae and necrosis surrounded by oedema and inflammation. Crepitus is occasionally present and the skin may be anaesthetic.

Investigations

1. Gram stained smears and cultures show the mixed nature of the infection.
2. At surgery the finding of oedematous, dull gray fascia and subcutaneous tissue with visible thrombi in penetrating vessels, confirms the diagnosis.

Prevention

Adequate debridement of wounds, blood loss replacement and antibiotics for contaminated wounds are essential to prevent postoperative necrotizing fasciitis.

Treatment

1. Surgical treatment. Debridement, under anaesthesia, with removal of necrotic skin and fascia is essential. The muscles are usually healthy and the limb can be salvaged.
2. Antibiotics. Penicillin 20-40 million units/day intravenously together with gentamycin (5 mg/kg/day) or Amikacin (15 mg/kg/day) are essential to control the mixed infection.
3. Blood transfusion, nutritional support and control of diabetes if present.

Prognosis

In many cases the diagnosis is missed. In elderly patients, the disease is likely to have a fatal outcome.

Chronic specific infections

Tuberculosis

The disease is still common in poor underdeveloped countries. In the developed countries, the recent rising incidence of "Acquired Immunodeficiency Syndrome (AIDS)" was accompanied by increased incidence of tuberculosis among those affected. Even well to do individuals can get the disease through ingestion of infected milk.

Aetiology

Organisms. Tuberculosis is caused by Gram-positive acid-fast rods (bacilli). There are two types of tubercle bacillus, the bovine and the human. The bovine bacillus is carried in infected milk. The human bacillus is now the commonest cause of tuberculosis in man.

Mode of infection

1. Inhalation. Direct aspiration of infected droplets from a patient suffering from open pulmonary tuberculosis is the commonest mode of infection.

2. Ingestion. The organisms are swallowed in contaminated milk from infected cows.
3. Direct inoculation. Rarely, the infection occurs by direct access through a wound or abrasion. In children a cold abscess may follow BOG vaccination.

Pathology

The characteristic lesion of tuberculosis is the tubercle.

Microscopic picture (Fig. 7.8)

The tubercle consists of a clump of bacilli surrounded by

- Epithelioid cells occupy the central part. They are oval or spindle shaped with faintly staining nuclei and abundant clear cytoplasm.
- Lymphocytes. The lymphocytes are arranged in a circular zone near the periphery.
- Langhan's giant cells. These are large cells of irregular shape that contain numerous small nuclei situated near one edge of the cell and are often arranged in a horse-shoe manner.

Fate of the tubercle

1. Caseation. This is a form of coagulative necrosis affecting the epithelioid cells near the center of the tubercle. As the tubercle enlarges, areas of caseation coalesce and may lead to the formation of a cold abscess (Fig. 7.9).
2. Fibrosis. When the host's resistance is high, the tubercle is surrounded by fibrous tissue and undergoes calcification. **Hypertrophic tuberculosis** Here there is overgrowth of granulation tissue with scanty giant cells and no caseation. The commonest example is ileocaecal tuberculosis.

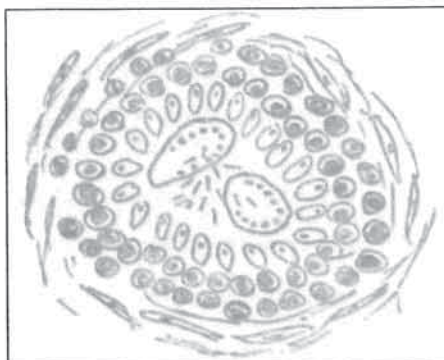


Fig. (7.8): Microscopic picture of tubercle



Fig. (7.9): The neck is a common site for a cold abscess

Spread

From the site of the original pathology, the disease spreads in the following ways

1. Direct extension to adjacent structures.
2. Lymphatic spread to regional lymph nodes and then to other lymph node groups or other tissues, e.g., tabes mesenterica from intestinal tuberculosis.
3. Via natural passages such as in the urinary tract, air passages and bowel.
4. Haematogenous spread leading to distant foci or miliary tuberculosis.

Clinical features

Tuberculosis usually affects children and young adults. It is commoner among the poorer classes where malnutrition, overcrowding, defective hygiene and poor general resistance predispose to the disease.

Generally

Manifestations of tuberculous toxæmia may be evident and include loss of appetite, loss of weight, night fever, night sweats and tachycardia.

Locally

Local manifestations depend on the affected site. The following lesions are characteristic of tuberculosis

1. Cold abscess. This forms a soft fluctuating mass that lacks the signs of acute inflammation unless there is secondary pyogenic infection. Aspiration reveals tuberculous pus (caseous material), although called cold abscess it is actually not cold, and not an abscess. It is slightly warm and it contains caseous material.
2. Tuberculous ulcer. This has an irregular outline, undermined cyanotic edge and deficient anaemic granulation tissue in the floor.
3. Tuberculous sinuses. These usually follow rupture of deep abscesses. They are usually multiple and unrounded by tuberculous ulcers and unhealthy scars.

Investigations

The following is a general outline to the diagnostic tests used in tuberculosis, additional tests are used according to site

1. Bacteriological examination of exudates, discharges and body fluids for tubercle bacilli. This includes staining techniques, e.g. Zeihl-Neelsen, culture on such media as Dorset's egg medium and guinea pig inoculation test.
2. Polymerase chain reaction (PCR) is an accurate and fast test, but is not widely available yet. It can detect even the smallest amount of specific DNA of the organism in tissue samples or discharge.
3. Tuberculin skin tests. A negative result excludes tuberculosis, but a positive result is of little significance.
4. Erythrocyte sedimentation rate (ESR) is increased but is not a specific test.
5. X-ray examination, e.g., of the lungs, bones, and kidneys may show characteristic changes.
6. Biopsy for histopathological examination may be needed to establish the diagnosis.

Treatment

General

1. **Chemotherapy.** It is always recommended to prescribe a combination therapy of at least two drugs. The commonly used drugs now are isonicotinic acid hydrazide (INH), rifampicin, pyrazinamide and ethambutol. Streptomycin is less commonly used because of its toxicity. Antituberculous drugs have some toxicity. Rifampicin is hepatotoxic, INH may cause peripheral neuritis, and ethambutol may produce visual impairment. Treatment should extend for at least nine months.
2. **Improving the general health.** Tonics, vitamins, good diet and open air are needed. Patients with open pulmonary tuberculosis need sanatorial treatment.

Local

Local management depends on the site affected, e.g. repeated aspiration for cold abscesses. For aspiration of a cold abscess the needle is introduced:

- In a non-dependent area
- Through healthy skin
- In a valvular manner
- Under aseptic precautions

Syphilis

Syphilis is a sexually transmitted disease. Its incidence has markedly decreased in the second half of the twentieth century as a result of effective antimicrobial therapy. Yet, the last decade or two have witnessed another increase in its incidence caused by promiscuous sexual practices.

Aetiology

- **Organism.** The spirochete, *Treponema pallidum*.
- **Mode of infection.** Acquired syphilis is contracted through intimate sexual contact with an infected person. In congenital syphilis the foetus is infected by transplacental spread in the later stages of pregnancy.

Clinical features of importance to the surgeon

- **Primary syphilis**
 - Genital, lip, and tongue ulcers (chancre)
 - Enlarged draining lymph nodes
- **Secondary syphilis**
 - Generalized lymphadenopathy.
- **Tertiary syphilis**
 - This stage usually occurs after 2 years and is characterized by the formation of localized gumma or diffuse infiltration. A gumma is a firm rounded mass which enlarges slowly and softens at the centre. When superficial, it ulcerates giving a characteristic gummatous ulcer. The ulcer has a circular outline, punched out edge, indurated base and a "wash leather" slough at the floor. Separation of the slough leaves a deep ulcer with bright red granulations. The ulcers tend to heal and breakdown again. Localized gumma may occur in the skin, bones, muscles, heart, liver and testis.

Investigations

1. Dark ground illumination to demonstrate spirochaetes in exudates and body fluids.
2. Immunological tests are either non specific or specific
 - Non specific tests Wasserman reaction, Kahn test and VDRL. False positive results may occur with malaria, glandular fever, leprosy, collagen diseases and chronic liver disorders.
 - Specific tests *Treponema pallidum* haemagglutination assay test (TPHA).

Treatment

Penicillin is the drug of choice. Penicillin G, 2-4 million units intramuscularly, is given as a single injection. Other antibiotics such as erythromycin and tetracycline may be used in penicillin-allergic patients. Erythromycin is given in a dose of 500 mg orally 4 times daily for 15 days.

Parasitic infections

Schistosomiasis (Bilharziasis)

Schistosomiasis is an endemic parasitic disease common among the rural population in Egypt.

Aetiology

Schistosomiasis is caused by a trematode worm which inhabits the venous system in man and affects the genitourinary organs or the intestinal tract. Two species are common in Egypt; *Schistosoma haematobium*, which gives rise to genitourinary lesions and *Schistosoma mansoni*, which gives rise to intestinal bilharziasis.

The parasite has two hosts, the definitive host is man and the intermediate host is a fresh water snail. The ova are discharged in the excreta of the definitive host (man). They hatch in water releasing the miracidia. This, in turn, finds its way to the intermediate host (snail), passes through different stages ending in the release of the cercariae. These escape from the snail and search for the definitive host (man), penetrate the skin of bare feet and legs during immersion or rarely the buccopharyngeal mucosa during drinking. They migrate in the tissue spaces, penetrate the lymphatics or venules and reach the right heart. They pass through the pulmonary circulation to the left heart and then to the systemic circulation. Only the cercariae that reach the portal circulation and the liver sinuses survive and grow into mature worms.

Haematobium worms leave the liver at an earlier stage and find their way through anastomotic channels between the mesenteric and systemic veins to reach the urinary organs. Mansonii worms remain in the liver until full maturity, they remain confined to the mesenteric veins and affect the bowel only. The worms migrate to the periphery to lay the eggs and then return back to the liver.

Pathology, complications and clinical features

These depend on whether the affection is mainly genitourinary or intestinal and will be discussed in details in respective chapters.

Investigations

1. Demonstration of ova in urine or stools.
2. Blood picture shows eosinophilia in early cases, and anaemia later.
3. Cystoscopy and sigmoidoscopy to visualize the lesions and obtain biopsy.
4. Radiological examination for specific lesions, e.g. Barium enema to show bilharzial polyps, ulcers, and pyelography to show bilharzial stricture of the ureters, and bladder neck.
5. Complement fixation and intradermal reaction tests are of little clinical applications.
6. Fine needle biopsy from the liver to show bilharzial periportal fibrosis and to differentiate it from cirrhosis.

Treatment

1. Preventive measures. Health education, provision of clean drinking water and sewage disposal systems are essential for prevention of the disease and to avoid reinfection after treatment.
2. Antibilharzial drugs. These should be given early before complications set in. The commonly used drugs include
 - a. Praziquantel (Biltricide^R) is given as a single oral dose of 40 mg/kg. No serious toxic effects are reported. It combines effectiveness, low toxicity and broad spectrum activity against both types of Schistosomiasis.
 - b. Oxamniquine (Vansil^R). This is a quinoline drug that is effective against *S. mansoni* infections only. It is given in a dose of 15-60 mg/kg daily for 1-3 days. Toxicity is insignificant.
 - c. Metrifonate (Bilarcil^R). This is an organophosphorus compound that is effective only against *S. haematobium* infections. It is given by mouth in 3 doses of 7.5 mg/kg at intervals of 2-4 weeks. Toxicity is negligible, but it should be used with caution in patients likely to be frequently exposed to organophosphorus insecticides as it reduces plasma concentrations of cholinesterase.

Filariasis

Aetiology

This is a tropical disease caused by a nematode worm, *Wucheraria bancrofti*, which lives in the main lymphatics of the abdomen and pelvis. Reproduction takes place there. The embryos (microfilariae) are extruded into the blood stream. The disease is transmitted by the bite of female mosquitoes of the *Culex* species through which microfilariae reach the lymphatic circulation where they grow to adult male and female worms. The disease occurs in certain areas in Egypt e.g. Sharkia, Guiza and Imbaba.

Pathology

The worms produce an intense inflammatory reaction which ends in obstruction of the big lymphatics. Lymph stasis predisposes to secondary streptococcal infection with recurrent attacks of lymphangitis which leads to further lymphatic obstruction.

Clinical features

The manifestations depend on the stage of the disease.

- In the early stages where lymphangitis is predominant, there is fever, rigors, malaise and pain in the affected limb. The regional lymph nodes are enlarged and may proceed to suppuration due to secondary infection. Affection of the lymphatics of the spermatic cord leads to funiculitis with diffuse matting of the cord structures.
- Later on, when obstruction of lymphatics sets in, distal lymphoedema becomes apparent with progressive thickening and hypertrophy of the skin and subcutaneous tissue (elephantiasis).
- Other manifestations of lymphatic obstruction depend on the site of obstruction e.g. chylothorax, chylous ascites, chylocele, and chyluria.

Investigations

1. Demonstration of microfilariae in thick blood films taken at night. A negative result is not against the diagnosis as microfilariae are not likely to be found in cases of less than 1-2 years duration. Furthermore, they may be prevented from reaching the blood stream by fibrosis of the lymphatics.
2. Examination of aspirated fluids may reveal microfilariae.
3. Biopsy of excised lymph nodes to detect adult worms. This is positive only in a minority of cases.

Treatment

1. Diethyl carbamazine (Banocide^R) is the treatment of choice.
2. Antibiotics are administered to control superadded streptococcal infection.
3. Specific measures vary according to the pathology, e.g. in lymphoedema, chyluria, and chylothorax.

Amoebiasis

Amoebiasis is a protozoal disease caused by *Entamoeba histolytica*. The parasite exists in two forms; the trophozoite is the active vegetative form that inhabits the colon, usually without causing symptoms, and the more resistant cystic form that is passed in stools. Invasion by trophozoites produces disease mainly in the colon and liver.

Clinical features

The disease is endemic in many parts of the world. The clinical picture depends on the site which is maximally affected.

Intestinal amoebiasis

Amoeba invade the colonic mucosa producing ulcers and colitis of varying severity.

- Amoebic dysentery. This is characterized by diarrhoea with blood-stained mucus, and tenesmus. The general condition is good. Tenderness may be elicited over the colon.
- Severe amoebic colitis, This is a severe form of the disease with fever and marked toxæmia. There is severe diarrhoea with blood and mucus resulting in dehydration. There is abdominal pain, cramps and tenesmus. The white cell count is markedly elevated. Untreated, the condition may proceed to perforation and peritonitis. Diagnosis is established by demonstration of the trophozoite in stool. Sigmoidoscopy reveals the characteristic small white-capped amoebic ulcers. Large ulcerated areas resulting from confluence of smaller ulcers may occur. The ulcers have ragged, undermined edges with congestion of the intervening mucosa. The condition must be differentiated from ulcerative colitis as treatment is entirely different. The condition responds to amoebicides and tetracyclines.
- Localized intestinal disease. This affects the caecum, sigmoid and transverse colon in that order of frequency. It may lead to stricture formation or a granulomatous mass called amoeboma. There is usually pain in the right lower quadrant associated with tenderness over the caecum and ascending colon. Stool analysis may reveal trophozoites and barium enema may show stricture. A history of dysentery is helpful in the diagnosis. Most cases respond to drug therapy. Strictures may need resection.

Hepatic amoebiasis (Chapter 31)

Echinococcosis (Hydatid disease)

Hydatid cysts affect the liver, and less commonly the spleen and lungs. Rarely the cysts may be found in the bones and brain. The disease is discussed in chapter 31.

Fungal infections

Candidiasis

Candida albicans and various other *candida* species are capable of producing both local and disseminated infection in hospitalized surgical patients. Overgrowth of *candida* occurs at mucocutaneous surfaces and in the intestinal tract of immunosuppressed and diabetic patients and in patients receiving broad spectrum anti-bacterial agents especially for long duration. Symptoms vary from those of oral lesions (red spots on the mucous membrane that soon become covered with whitish membrane) to dysphagia (candidiasis of oesophagus) or diarrhea and other digestive disturbances.

Treatment

1. Oral non absorbable antifungal agents such as nystatin are useful in oral candidiasis.
2. Systemic antifungal therapy is required for severe infections. The commonly used drugs include amphotericin B, 5-fluorocytosine and fluconazole.

Antibiotics in surgical infections

Choice of the suitable antibiotic

Before selecting an antibiotic for the treatment of an infection, the surgeon must consider two factors; the patient and the known or likely causative organism. Factors related to the patient which must be considered include history of allergy to antibiotics, renal and hepatic function, resistance of the host, ability to tolerate oral drugs, race, age and, if female, whether pregnant, lactating or taking an oral contraceptive. These factors, together with the antibiotic sensitivity of the known or possible organism, guide the choice of antibiotics.

The following guidelines are helpful whenever prescribing an antibiotic

1. An initial diagnosis is essential before starting antibiotic therapy. Relevant samples, e.g., urine, sputum, pus etc should be obtained for culture and sensitivity testing before the first dose of antibiotic is given.
2. The initially chosen antibiotic should be effective against the most likely pathogen. Toxic agents or extremely costly agents are better avoided if an equally effective substitute exists.
3. The length of antibiotic course should be judged based on the pathology, and the clinical and laboratory assessment. Prolonged antibiotic courses should be avoided, whenever possible.
4. The change from one antibiotic to another should be based on culture and sensitivity tests in association with clinical response. If there is a satisfactory clinical response, we do not change antibiotics just for the sake of culture and sensitivity results.

Antibiotics in common use

The following is a brief outline of the commonly used antibiotic groups. For further details the reader is encouraged to refer to appropriate text books.

1. **B-Lactam antibiotics.** Antibiotics in this group exert their activity through the similarity of the B-lactam ring to the peptidoglycan that forms part of the bacterial cell wall in both Gram-positive and Gram-negative bacteria. They lead to inhibition of cell wall synthesis, and hence, cell lysis. Three classes of antibiotics belong to this group
 - **Penicillins**
 - Penicillin G is the prototype of the whole group and is effective mainly against Gram-positive bacteria such as streptococci and spirochaetes.
 - Semisynthetic penicillins (e.g., methicillin) exert a wider activity against streptococcus and staphylococcus species.
 - Ampicillin lacks activity against some staphylococci but is active against enterococci and some Gram-negative bacteria.
 - Combining amoxycillin with a lactamase inhibitor (e.g., clavulanate) serves to extend the spectrum of activity against Gram-positive, Gram-negative aerobic and some anaerobic organisms.
 - **Cephalosporins.** Three generations of cephalosporins have been described (first, second and third)
 - **First generation cephalosporins**, e.g. cephazolin and cephalexin, exert excellent activity against Gram-positive bacteria such as common strains of Staph. aureus. However, staphylococcal strains that are resistant to methicillin are invariably resistant to first generation cephalosporins.
 - **Second generation cephalosporins** e.g. cefoxitin and cefuroxime, have a slightly narrower Gram-positive bacterial spectrum but a much better Gram-negative aerobic activity. Some agents in this group also exert activity against anaerobic organisms, e.g. cefoxitin.
 - **Third generation cephalosporins** are excellent anti-Gram-negative aerobic agents but their Gram-positive activity is variable. This group includes ceftriaxone, ceftazidime, and cefotaxime.
 - **Carbapenems.** These agents have wide spectrum that includes Gram-positive bacteria, most Gram-negative aerobes including pseudomonas, and anaerobes. Imipenem, meropenem and aztreonam are members of this group.

2. **Aminoglycosides.** Aminoglycosides are effective mainly against Gram-negative aerobic organisms with little activity against Gram-positive or anaerobic bacteria. They act by interfering with bacterial protein synthesis. Commonly used agents belonging to this group include gentamycin, tobramycin and amikacin. They all have considerable ototoxicity and nephrotoxicity. The use of these agents should therefore be limited and controlled by serum creatinine and drug level values. Safer alternatives e.g. third generation cephalosporins, carbapenems or quinolones, should always be considered first.
3. **Quinolones.** Quinolones exert activity against Gram-negative aerobic bacteria. Available agents include, norfloxacin, ciprofloxacin and levofloxacin. They are most commonly used in urinary tract infections.
4. **Glycopeptides.** Possess activity against Gram-positive bacteria and are more potent than most other agents. They interfere with bacterial cell wall synthesis. They are effective against methicillin resistant Staph. as well as anaerobic Gram-positive cocci. Vancomycin is the drug of choice for the treatment of antibiotic-induced colitis which is caused by overgrowth of clostridium difficile. Belonging to this group are vancomycin, linezolid, rifampicin, clindamycin and metronidazole.
5. **Tetracyclines and chloramphenicol** exhibit anaerobic activity but their use in surgical patients has been superseded by more effective drugs.
6. **Trimethoprim-sulphamethoxazole (Septrin)** is effective against a variety of Gram-negative aerobic bacteria and some unusual infections observed in the immunosuppressed patients such as those caused by *Pneumocystis carinii* and *Nocardia asteroides*.
7. **Macrolides** they exert activity against Gram-positive bacteria and are usually used in patients allergic to B-lactam agents. This group includes erythromycin, azithromycin and clarithromycin.

Complications of Antibiotics

1. **Hypersensitivity reactions.** These are especially common with penicillin and streptomycin. They include urticaria, fever, angioneurotic oedema, asthma and rarely fatal anaphylactic reactions.
2. **Vitamin B deficiency** is due to alteration of bowel flora especially with prolonged use of antibiotics.
3. **Superinfections** are caused by resistant strains such as proteus, pseudomonas or candida. Such infections may affect the mouth, bowel, urinary tract or lung and are very difficult to control.
4. **Specific toxicities,** e.g. nephrotoxicity and ototoxicity are noted with aminoglycosides, marrow toxicity with chloramphenicol, while dental changes are caused by tetracyclines in children.

Table (7.1) Illustrates the activity of various antibiotics (remarks + means reliable activity)

Antibiotics	Staph. aureus	Enterococci	E. coli	Pseudomonas	Anaerobes
Penicillin/beta lactamase inhibitor					
- Ampicillin/sulbactam	+	+	+	-	+
- Amoxycillin/clavulinate	+	+	+	+	+
1st generation cephalosporins					
- Cephazolin	+	-	+	-	-
- Cephalexin	+	-	+	-	-
2nd generation cephalosporins					
- Cefoxitin	+	-	+	-	-
- Cefuroxime	+	-	+	-	-
3rd generation cephalosporins					
- Ceftriaxone	+	-	+	+	-
- Ceftazidime	+	-	+	+	-
- Cefotaxime	+	-	+	+	-
Carbapenems					
- Imipenem-cilastatin	+	+/-	+	+	+
- Meropenem	+	+/-	+	+	+
- Aztreonam	+	+/-	+	+	+
Aminoglycosides					
- Gentamicin	+	+	+	+	-
- Tobramycin	+	+	+	+	-
- Amikacin	+	+	+	+	-
Quinolones					
- Ciprofloxacin	+/-	-	+	+	-
- Levofloxacin	+/-	-	+	+	-
- Norfloxacin	+/-	-	+	+	-
Glycopeptides					
- Vancomycin	+	+	-	-	-
- Linezolid	+	+	-	-	-
- Rifampicin	+	+	-	-	-
- Clindamycin	+	+	-	-	+
- Metronidazole	-	-	-	-	+
Macrolides					
- Erythromycin	+	-	-	-	-
- Azithromycin	+	-	-	-	-
- Clarithromycin	+	-	-	-	-

Decontamination

It is the process of removing microbial contaminants. It can be carried out by one of three methods, cleaning, disinfection or sterilization.

- (A) **Cleaning** it is the process that removes visible contamination. It is a necessary prerequisite for effective disinfection or sterilization.
- (B) **Disinfection** it is the process that reduces the number of viable micro-organisms to acceptable levels. A topical disinfectant that may be safely applied to epithelial tissues is known as an antiseptic. Disinfection can be achieved by moist heat as boiling water. Chemical disinfection is used when the use of heat is not appropriate. Glutaraldehyde (Cidex) is a common example, immersion of endoscopes or laparoscopes achieves control of bacteria, HCV and HIV. The equipment should then be washed with sterile saline prior to its use as the agent is toxic (**Table 7.2**) illustrates the commonly used antiseptics.

Table (7.2): The commonly used antiseptics

Name	Presentation	Uses
Chlorhexidine (Hibiscrub)	Alcoholic 0.5%, aqueous 4%	Skin preparation
Povidone iodine	Alcoholic 10%, aqueous 7.5%	Skin preparation, fast acting hand wash and open wounds. Effective against Gram-positive and Gram-negative organisms
Alcohols	70% ethyl alcohol	Skin preparation
Hexachlorophene	Aqueous bisphenol	Skin preparation, hand washing Effective against Gram-positive organisms

(C) **Sterilization.** It is the process of complete destruction of all viable micro-organisms including spores, viruses and mycobacteria. The standard method of sterilization is the steam under pressure (autoclaving) which attains 134 °C at 2 atmospheres. The time of sterilization is 20-30 minutes.

Other methods of sterilization

- Ethylene oxide (EO) gas for wrapped and unwrapped heat-sensitive equipment.
- Glutaraldehyde at longer immersion times.
- Gamma rays for sterilization of catheters, syringes or delicate equipment on industrial basis.
- Gas plasma for delicate catheters and endovascular wires at hospital.

When operating on patients with HCV or HIV

- Clear documentation in the patient's records.
- Wear a full-face high quality mask.
- Eye protection.
- Use disposable water proof gowns.
- Wear double gloves.
- Let the scrub nurse handle sharps as the scalpels to the surgeons indirectly in a kidney dish.

The discipline in the operating theater is important in preventing infections

- Air-borne bacteria are a source of postoperative sepsis. Use special air filters, the theatre ventilated in a way that air is driven in layers (laminar flow) or under positive pressure.
- Limit number of people and their movements.
- There should be a red line beyond which all staff should wear face masks, head caps and clean shoes.

BURNS AND PRINCIPLES OF PLASTIC SURGERY

Introduction

A burn is one of the most serious injuries that can be inflicted on the body. Burns cause dramatic damage to the physiological functions of the skin, and can lead to serious local and systemic complications that endanger the patient's life. Furthermore, burn survivors commonly develop crippling and unsightly scars. The modern management of burns requires specialized care and enormous financial costs.

By size and weight, the skin is the second largest organ in the body next only to the muscles. Loss of large amounts of skin, if not replaced, is incompatible with life. The skin has limited ability of regeneration and lacks much of the functional reserves of internal organs.

Histology of the Skin

The skin is formed of two layers epidermis and dermis (Fig. 8.1). The epidermis, is formed of stratified squamous epithelium. The dermis is formed of connective tissue. The skin appendages (hair follicles, sweat glands, and sebaceous glands) arise from the epidermis and grow down into the dermis. When the epidermis is damaged in burns, epithelium from these appendages can creep up to cover the denuded area.

CHAPTER CONTENTS
▪ Introduction
▪ Aetiology of burns
▪ Pathology of burns
○ Extent and depth
○ Histopathology
○ Pathophysiology
○ Complications
▪ Management of burns
○ First aid
○ Admission to hospital
○ Resuscitative fluid therapy
○ Local burn wound care
▪ Special types of burns
▪ Prognosis of burns
▪ Principles of skin coverage
○ Grafts
○ Flaps
○ Tissue expanders
▪ Aesthetic surgery

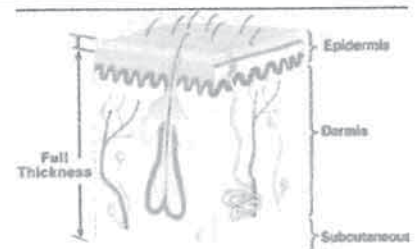


Fig. 8.1. Structure of skin.

Functions of the Skin

1. It forms a barrier against invasion by micro-organisms.
2. It prevents excessive loss of water from the body.
3. It helps in the regulation of body temperature by secretion of sweat.
4. Sensory receptors in the skin protect the body from injurious insults.
5. Formation of vitamin D.

Aetiology of burns

Burns can result from a variety of thermal injuries

- Scalds are caused by boiled liquids. They often affect children.
- Flame burns can affect any age and are often due to ignition of clothing and is the most common burn industrial accidents.
- Electric burns are usually due to contact with an electric socket or a low voltage battery. Contact with high voltage may be fatal since the blood and bones are good conductors, thus eventually every organ in the body may be affected.

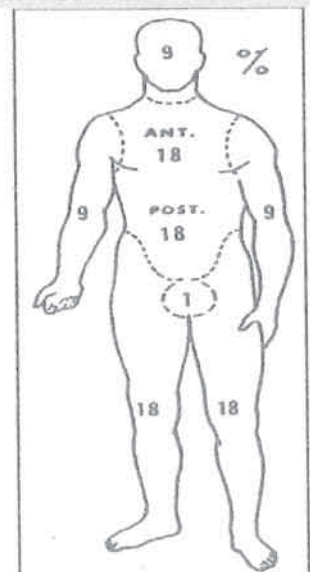


Fig. (8.2) Rule of nines

thus eventually every organ in the body may be affected.

- Chemical burns are due to either acids or alkalies.
- Inhalation and respiratory burns are due to exposure to hot gases and may affect the upper or lower airway.

Pathology of burns

Extent and depth of burns

1. The **extent** is the percent of burnt skin surface area in relation to the whole body surface area. This follows the rule of 9 (Fig. 8.2). In children the rule of 9 needs some modification due to the large size of the head in comparison to the rest of the body. According to the extent burns are classified into
 - A major burn involves more than 30% of the body surface area.
 - An intermediate burn involves 15-30% in adults and between 10-30% in children.
 - A minor burn is less than 15% in adults and 10% in children.



Fig. 8.3. Sunburn.

2. Depth

- First degree burns. The usual example is sunburns (Fig. 8.3). Only the epidermis is damaged causing erythema of the skin. They heal rapidly.
- Second degree burns. The epidermis and a portion of the dermis are damaged. Provided that no infection occurs, epithelial regeneration can occur from the remnants of hair follicles and sweat glands in the dermis and the burn will heal in 3 weeks. If infection occurs, these epithelial elements are destroyed and the case will be changed to a full thickness burn. This degree is further subdivided into
 - i. Superficial partial thickness burns (Fig. 8.4). These usually heal with epithelialization in 10-14 days.
 - ii. Deep partial thickness burns (Fig. 8.4). These are slower to heal (25-35 days). Cells from the residual uninjured epithelium of deep sweat glands and hair follicles creep up to cover the surface.
- **Third degree (full thickness) burns.** There is complete destruction of the epidermis and dermis (Fig. 8.5). Epithelial coverage can occur only by epithelial migration from the edges of the burnt area which is a very slow process. After separation of the eschar by the third week, the patient should be prepared for skin grafting.



Fig. 8.4. Superficial partial thickness burn by boiling water



Fig. 8.5. Deep (third degree) burn.

The differentiation of partial thickness from full thickness burn is sometimes difficult and even in the same area, both patterns may intermingle together. However, the following may help to differentiate.

Appearance Partial thickness burns are characterized by the formation of blisters surrounded by erythema. Their surface is mottled red and is usually moist due to exudation of plasma. In full thickness burns, the area is dry. White or black eschar (burn slough) is present with possible visible thrombosed subcutaneous vessels.

Presence of pain Partial thickness burns are very painful and sensitive to air, while full thickness burns are painless due to loss of the terminal nerve endings. This can be elicited clinically by the pinprick test.

Rate of healing In mixed areas, partial thickness burns should heal within 3 weeks, while in full thickness burns granulation tissue formation and eschar separation start to occur after 3 weeks.

Histopathology

Human skin is injured by heat in two ways. An immediate direct cellular injury occurs first and then a delayed injury is inflicted as a result of progressive dermal ischaemia. The degree of tissue destruction is related to both temperature and duration of exposure to the heat source. Cell damage is essentially due to protein denaturation and many of these changes are reversible. However, at temperatures greater than 45°C, protein denaturation exceeds the capacity of cellular repair. Histologically, thermal injury to the skin results in 3 distinct zones of trauma (Fig. 8.6).

- **The central inner zone (zone of coagulation).** This forms the inner layer of the visible burn eschar. If left, the eschar separates within 3 weeks leaving either a bed of granulation tissue (in full thickness burns) or a re-epithelialized bed (in partial thickness burns).
- **The intermediate zone (zone of stasis).** This area surrounds the zone of coagulation. It contains viable tissues that may die over the next 48 hours post-burn, if tissue oxygenation and adequate nutrition are not maintained.
- **The outer zone (zone of hyperaemia).** This area contains inflammatory mediators (prostaglandins, histamine and kinins), which contribute to the formation of tissue oedema. Tissues in this zone normally recover within 7-10 days unless subjected to infection.

The rate of burn healing depends on the density of the surviving adnexal epithelial structures within the dermis, especially hair follicles and sweat glands which are present deep to the epidermis. In a partial thickness burn, healing should be achieved by epithelialization within 3 weeks-without any surgical intervention (Fig. 8.7), while in a full thickness burn, separation of eschar from living tissue occurs at about 4 weeks leaving clear granulation tissues. If left without grafting healing occurs by fibrosis and scarring (Fig. 8-8).

Pathophysiology

As mentioned before the skin plays a vital role in prevention of excessive loss of water and consequently calories from the body. It is also a good barrier against invasion by microorganisms. The following pathological sequelae occur in the burnt patient

1. Increased capillary permeability in the burnt area leads to the loss of enormous amounts of fluids and proteins in the damaged area. This is maximum in the first 8 hours and continues for 48 hours.
2. Excessive loss of water by evaporation through the burnt skin takes place. A patient with a full thickness burn can lose up to 200 ml/m² hour. For every litre of water evaporated, the body loses 560 KCal. The result will be severe dehydration and catabolism.

3. Increased metabolic rate and nutritional demand.
4. Immunosuppression. Both humoral and cell-mediated immune systems are affected. The burnt area is colonized by bacteria. It is impossible to eradicate these bacteria except after skin coverage.

Complications

1. Systemic complications

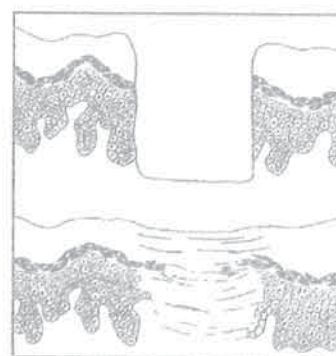
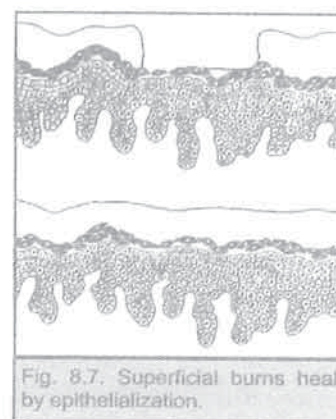
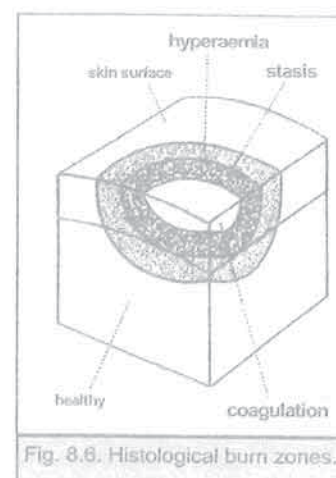
As stressed before, in major burns all body systems may be affected and serious complications may happen.

- (a) Inhalation injury. Asphyxia may occur immediately from inhalation of heat or noxious gases. The result is bronchospasm and later progressive hypoxia and hypercarbia. Proper oxygenation is essential to avoid such complications. Additionally, atelectasis, pneumonia, pulmonary emboli, emphysema and pulmonary oedema may follow. Finally respiratory failure may occur with systemic sepsis.
- (b) Neurogenic shock is caused by pain.
- (c) Hypovolaemic shock may develop if the fluid losses are not properly corrected.
- (d) Renal failure. Acute renal failure can occur after prolonged uncorrected hypovolaemia.
- (e) Gastro-intestinal complications. Acute ulceration of stomach and duodenum (Curling's ulcer) are rarely seen nowadays with the routine prophylactic use of antacids and H_2 receptor antagonists during the initial burn management. Ileus usually occurs during the early post-burn period and may necessitate the insertion of a nasogastric tube. Recently, acute ulcerations of the colon are reported and may be deep reaching the serosa. It is suggested to be secondary to fungal infection.
- (f) Multiple organ failure may follow major burns particularly with sepsis (refer to SIRS in chapter 6). The lungs, heart, kidney, liver and the nervous system may be affected. The outcome is usually poor.

2. Local complications

(a) Early local complications

- Infection is the primary cause of death in burnt patients. It may be bacterial, viral or fungal, and can occur from both exogenous or endogenous sources. Infection may lead to the development of septicaemia and septic shock. It occurs usually between 4-7 days postburn. Treatment is essentially by prevention through proper local burn wound care. The value of systemic antibiotics in the prevention of infection is doubtful.
- In deep circumferential burns of the limbs and chest, constricting eschars may form and should be treated by urgent escharotomy, i.e., release of constricting eschars.



- In burns of the face and neck, oedema may lead to suffocation and urgent tracheostomy may be needed.
- Compartment syndrome

(b) Delayed local complications

- Contractures across joints (Fig 8.9).
- Scar formation (hypertrophic or keloid).
- Malignant transformation (Marjolin's ulcer) in long-standing unstable scars is rare.

Management of Burns

This needs the utmost care and attention, and it is better to treat patients with major burns in special centers.

First aid

1. A patent airway should be assured. If there is airway obstruction, an endotracheal tube should be inserted.
2. A strong analgesic as 50 mg pethidine is administered IV. Intramuscular injections are avoided as absorption is poor. Analgesic administration is repeated as needed.
3. Tetanus prophylaxis.
4. Saline or tap water, at room temperature, can be poured over the burnt area for 15 minutes to limit the depth of burn, decrease oedema and relieve pain. Using ice-cold water from a refrigerator is contraindicated as it may induce more tissue damage.

Admission to hospital

Minor burns (less than 15% in adults and 10% in children) can be treated as outpatients. In such cases the treatment consists of dressing using the proper local chemotherapeutic (mentioned later), and analgesia.

Indications of the admission to the hospital

1. Inhalation injury.
2. Burn size over 15% in adults or 10% in children.
3. Any full thickness burn.
4. Burn in association with trauma or comorbidity.
5. Electric burns.
6. Chemical burns.

If it is decided to admit the patient, the following is performed

- A wide bore IV. cannula is inserted rapidly before the veins get collapsed.
- A Foley urethral catheter is introduced to check urine output.
- Treatment essentially consists of fluid therapy to compensate for the extensive losses and local care of the burn wound.
- The value of systemic antibiotics in prevention of burn wound infection is controversial.

Resuscitative fluid therapy

The amount and rate of fluid replacement are determined by the weight of the patient and the percentage of the total body surface area injured. It is essentially given for the first 48 hours and after that according to the response of the patient a maintenance therapy may continue. Several formulae have been proposed for estimating the patient's fluid needs, keeping in mind that the greatest loss of fluids occurs during the first 8 hours post-burn and continues more slowly over the next 16 hours.

Parkland's formula is commonly prescribed. It is estimated as follows (Fig. 8.10):

- First 24 hours: 4 ml/Kg/1% surface area as lactated Ringer's solution. Half the calculated amount is administered in the first 8 hours and other half over the next 16 hours.
- Over the next 24 hours half the previous amount is administered.
- Administration of blood is usually needed in major deep burns. It can be started after 48 hours, guided by the haematocrit value.
- It is to be noted

It is to be noted that in all formulae, the maximum percentage of burn calculated is 50%, otherwise serious overinfusion may occur. Oral intake is avoided during the first 48 hours to avoid gastrointestinal complications and is started gradually after that.

Monitoring

The adequacy of intravenous resuscitation is judged by

1. Regular check-up of vital signs.
2. Urine output should be 0.5-1 ml/kg/hour.
3. C.V.P. in critical cases.

The nutritional status

Of the patient should not be neglected. Patients who have extensive burns are liable to have a serious catabolic status due to the combined effects of anorexia, extensive water and consequently caloric losses and due to sepsis if present. Introduction of intravenous hyperalimentation (Chapter 9) has made it easy to correct this problem and to support the patient nutritionally during this critical period.

Local burn wound care

Early care

After general resuscitative measures have been started, attention should be directed to the burn wound.

1. **Escharotomy.** Constricting eschars (in the limbs and chest) may have to be released immediately (Fig. 8.11). Sometimes, fasciotomy (in deeper burns) may be limb saving and has to be done as a first aid measure.
2. **Cleansing,** removing loose skin, and initial conservative debridement. The aim of the local wound care is to avoid infection.
3. **Topical antimicrobial** agents should be applied. The ideal topical cream should be in a water soluble base, prevents dryness, not painful, nonallergenic, non toxic and most importantly bactericidal but not injurious to viable cells in the burn wound. The three most commonly used topical agents are; silver sulphadiazine, silver nitrate solution and mafenide acetate.
4. After application of the local cream, the wound is managed by either leaving it exposed (the exposure method) or by covering it by a bulky occlusive dressing (the occlusive method) that is changed every 2- 3 days depending on the state of the burn wound. Both the occlusive and exposure methods are equally effective. However the



Fig. 8.9. Contracted keloidal scar of the neck that followed spontaneous healing of a deep burn.

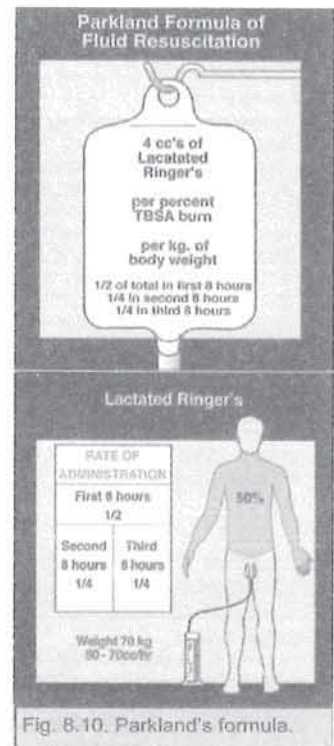


Fig. 8.10. Parkland's formula.

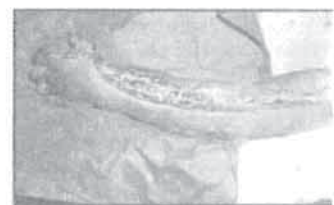


Fig. (8.11) Escharotomy for deep circumferential burns

exposure method requires isolation of the patient in completely aseptic atmosphere and following all aseptic rules by the personnel dealing with the patient.

Exposure method has the following advantages

- It is more comfortable to the patient.
 - It avoids the need for repeated change of dressings which implies a great burden on nursing staff and is rather expensive.
 - It inhibits the growth of bacteria by the dry air surrounding the burn.
- Exposure method is particularly indicated for
- Burns of the face, neck and perineum.
 - Burns involving one side of the trunk.

Later care

Following the previous regimen of wound care, all partial thickness burns should heal within 2-3 weeks, while full thickness burns will require closure by autogenous skin grafts. For deep burns

1. **Autologous skin grafting.** Partial thickness skin grafts (Thiersch grafts) are commonly used to cover the large raw areas produced by burns. A graft is applied after either spontaneous separation of eschar (3-4 weeks), or early burn wound excision. Early burn wound excision is the preferred method for deep burns of the hand. Patients are usually subjected to surgery 3-5 days post-burn (before bacterial infection sets in), where tangential excision of the damaged dermis is carried on. When healthy bleeding tissues are reached a skin graft is applied.
2. **Biological dressings.** In case where autografts are not enough, or the local wound conditions are not favourable, one may resort to biological dressings which when applied temporarily would render the wound less painful, minimize fluid and protein loss and control infection. Examples of biological dressing are allografts (cadaver's skin), xenograft skin (pig's skin) and amniotic membrane. More recently, artificial skin substitutes are used with promising results. All biological dressings are only applied after removal of the eschar and are changed every 3-4 days. It is to be stressed that biological dressings are only temporary and that permanent closure can be achieved only by autograft skin.
3. **Prevention of contractures.** Contraction is a normal component of wound healing and its prevention across joints can be achieved by proper splintage, immobilization in the best functioning position, and physiotherapy.
4. A modern trend is to apply synthesized dermis over the post burn granulating raw areas, then cover it with split thickness graft to have a better scar and to prevent contractures.

Special types of burns

Electric burns

This burn is divided into high and low tension injuries according to the voltage responsible for the injury (whether above or below 1000 volts). The tissue damage is due to the passage of the electric current through blood and bones. The affected blood vessels usually show late thrombosis with delayed progressive ischaemic necrosis at different sites. Clinically 3 types of skin damage may result from electric burns:

1. Contact burns occur at points of current entry and exist from the body (Fig. 8.12).

2. Burns from current exist and re-entry at adjacent parts.
3. Thermal burns from ignition of clothing due to the heat generated by the current passing through the body.

Inhalation burns (Fig. 8.13). Flames or very hot air may be inhaled causing burns in

- Upper respiratory tract causing laryngeal oedema and possibly stridor.
- Lower respiratory tract causing pneumonia and ARDS, which lead to respiratory failure.

Inhalation burns are suspected in the following circumstances

- Closed space burns.
- Facial burns.
- Burnt nasal hairs.
- Carbonaceous sputum
- Wheezing, pharyngeal oedema, or hoarseness of voice.

When inhalation burns are suspected consider:

- Early intubation.
- High-flow oxygen.
- Admission to hospital, with possible need for mechanical ventilation.

Chemical burns

Chemical agents burn by oxidation, reduction, corrosion or protoplasmic poisoning. The severity of a chemical burn is determined by the strength of the agent, its amount, duration of skin contact and its mechanism of action. Management of a chemical burn is both local and systemic. Systemic control is by administering the proper antidote in case the agent is absorbed. Locally, the agent should be thoroughly cleansed, all saturated clothes are removed and affected areas irrigated with huge amounts of water.

Cold injury

Is a form of thermal injury that is due to exposure to cold and depends upon the duration of exposure and surrounding temperature. The trauma leads to freezing of tissues and usually affects the hands, feet, ears and nose. Examples are frostbite, trench foot and chilblain.

Prognosis of burns

Burn factors

1. Extent. This is the most important factor. Mortality is about 50% if the extent of burn is 50%.
2. Depth. Deeper burns carry higher mortality and more disfigurement.
3. Site. Burns of the face are the worst because they are likely to be accompanied by inhalation burns of the respiratory tract.
4. Infection. Septicaemia is the major killer of burn victims.

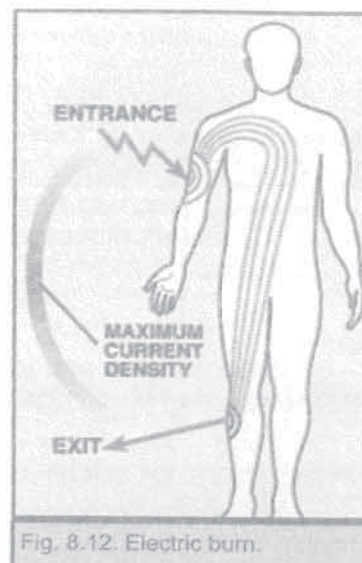


Fig. 8.12. Electric burn.

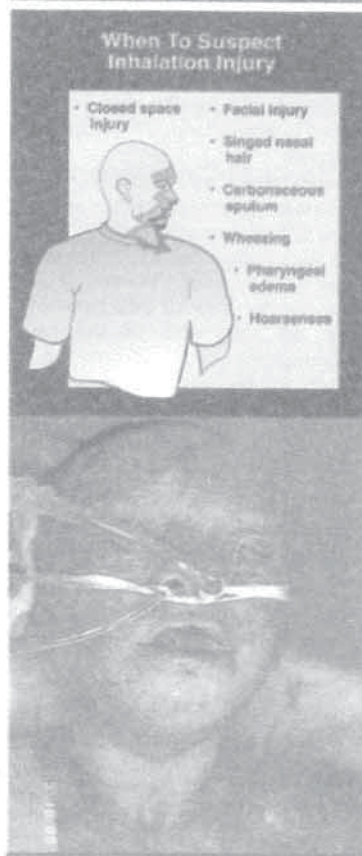


Fig. (8.13) Inhalation burns should be suspected with burns of the face. Early tracheal intubation is needed.

5. Type. High-voltage electric burns are the worst. They are usually fatal. Scalds with boiling water commonly cause superficial burns and are the least harmful.
6. Associated injuries.

Patient factors

1. Age. Burn victims at extremes of age (children and elderly) have bad prognosis.
2. Concomitant diseases, e.g., diabetes and coronary heart disease.

Treatment factors

Patients who are treated in specialized centres have better chance of survival and avoidance of complications.

Principles of skin coverage

Skin defects represent one of the common problems in surgical practice. These are the result of trauma, and burns, or follow the surgical resection of different benign and malignant lesions. Different types of grafts and flaps are in use, and it is the duty of the surgeon to choose the most suitable for each case.

Table (8.1) Comparison between the two types of skin grafts

	Split thickness (Thiersch) graft	Full thickness (Wolfe) graft
Advantages	<ul style="list-style-type: none"> ▪ Survive easily (good take) ▪ Donor site heals spontaneously 	<ul style="list-style-type: none"> ▪ Nearly all qualities of normal skin ▪ Minimal contraction ▪ Resistant to trauma ▪ Better sensation ▪ Aesthetically better
Disadvantages	<ul style="list-style-type: none"> ▪ Few qualities of normal skin. ▪ Marked contraction ▪ Weak resistance to trauma ▪ Poor sensation ▪ Aesthetically poor 	<ul style="list-style-type: none"> ▪ Less chance of survival (less take) ▪ Donor site must be closed surgically ▪ Donor sites are limited

1. Skin grafts

A skin graft involves tissue that is completely detached from its blood supply in the donor area and receives its new blood supply from the base of the wound, the recipient area.

Types

A segment of skin, including the epidermis and a variable thickness of the dermis, is totally separated from its blood supply in the donor area, and is then transplanted to the raw recipient area. There are two types of skin grafts, the split thickness and the full thickness varieties. The split thickness (Thiersch) graft includes the epidermis and part of the dermis. The full thickness (Wolfe) graft includes the epidermis and the full thickness of the dermis. Table 8.1 demonstrates the advantages and disadvantages of each.



Fig. 8.14. Split thickness graft.

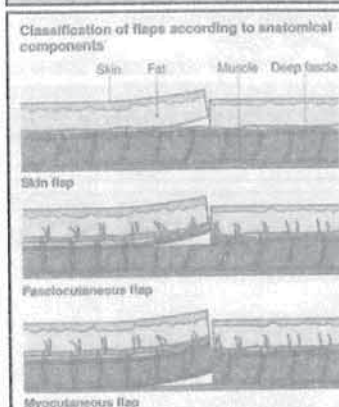


Fig. 8.15. anatomical components of flaps.



Fig. (8.16) Island flap based on superficial temporal vessels

Occasionally the skin graft may include an underlying tissue, e.g. subcutaneous tissue, muscle, cartilage, or bone. In such cases it is termed a composite graft.

Graft survival

For a skin graft to survive, it must obtain a blood supply from its recipient bed. Thus, the vascular status of the bed determines to a great extent the survival (take) of a skin graft. Poorly vascularized tissues such as bare cortical bone (devoid of periosteum), bare tendon, and bare cartilage will not take a skin graft, but can be covered by flaps. Skin grafts survive by new vessels that grow from the recipient bed to supply the graft. This neovascularization takes about 5-7 days to be established depending upon the status of the recipient bed, the graft thickness (the thinner the graft, the faster is the take), and the size of the graft (a small graft takes rapidly). During this period of neovascularization, the graft needs to be supported by some form of immobilization and mild compression.

Technique

Split thickness grafts are harvested by a special knife (dermatome) from hidden areas as the thighs. The donor site heals with complete epithelization within 3 weeks. The epithelium is derived from that of the skin appendages (hair follicles, sweat glands, and sebaceous glands) that have deep extensions into the dermis. Full thickness grafts can be harvested from anywhere in the body using the scalpel blade. Generally, they are taken from an area close to the recipient site for better colour matching, e.g. from above the clavicle or the postauricular area to cover facial defects.

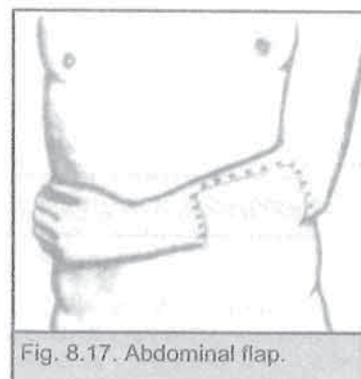


Fig. 8.17. Abdominal flap.

2. Flaps

The tissues to be transferred from one site of the body to another must maintain their blood supply for nourishment. There are different types of flaps (Fig. 8.15).

1. **Skin flaps** These can be classified into local and distant flaps.

a. Local flaps

- **Random pattern flaps** (Fig. 8.16). These flaps have no anatomically recognized blood supply. Thus, they should have a strict length to width ratio in order to survive. A safe random pattern flap has a length to width ratio of 2:1 maximally.
- **Axial pattern flaps**. These are supplied by a known artery, and have no limitations as regards length to width ratio. The whole area supplied by the specific cutaneous artery can be transferred safely. Additionally, adjacent nerves may be included, and the flap will be a sensory one. Axial flaps may retain their skin attachment to the donor area and are termed pedicled flaps. If they retain their blood supply without the skin attachment, they have a wider arc of rotation and are termed island flaps (Fig. 8.16).



Fig. 8.18. Cross leg flap.

- b. **Distant pedicle flaps**. These keep the recipient site attached to the donor site for a period of 2-3 weeks in order to allow for neovascularization, before separating the base. Examples are flaps from the abdomen or groin that are used to resurface the hand, (Fig. 8.17) and those from one leg to another (cross leg flap) (Fig. 8.18).

2. **Musculocutaneous (myocutaneous) flaps.** These are axial flaps that receive their blood supply from the underlying muscles through perforator vessels. The muscle is lifted from its bed with the overlying fascia and an area of skin, yet retaining the neurovascular pedicle of the muscle. The whole flap is then rotated in an arc to cover the raw area. The incorporation of the pedicled muscle in the flap enhances the survival of the cutaneous component. A famous example is the latissimus dorsi myocutaneous flap that is used to cover defects resulting from wide radical mastectomies (Fig. 8-19), and the pectoralis major myocutaneous flap used to cover defects in the neck or face.
3. **Fasciocutaneous flaps.** In an attempt to avoid the functional deficit of muscle transfer and the sometimes excess bulk of myocutaneous flaps, fasciocutaneous flaps were introduced. They have the same principles as the previous one, but their blood supply is through fasciocutaneous perforators.
4. **Microvascular free flaps** With the advent of microvascular techniques, it is now possible to anastomose vessels of less than 1 mm in diameter. Thus without the need for keeping the patient immobilized for 2-3 weeks, and the possible joint stiffness, it is now feasible to transfer an axial flap from one site to another distant site by anastomosing the artery and veins of the flap to the corresponding vessels in the recipient area.



Fig. (8.19) Latissimus dorsi myocutaneous flap

3. Tissue expanders

Tissue expanders are inflatable silicon implants. They are placed subcutaneously in collapsed state. Over several weeks the expander is gradually inflated with saline through a subcutaneous port. The overlying skin is gradually stretched to accommodate a larger area. Finally, the expanded skin can be used to cover defects and the tissue expander is removed (Fig. 8.20).

Fig. (8.21) represents the Reconstruction Ladder Starting from the most simple (bottom) to the most sophisticated method for closure of skin defects.



Fig. (8.20) Tissue expander. In this case it is used to cover a scalp defect.

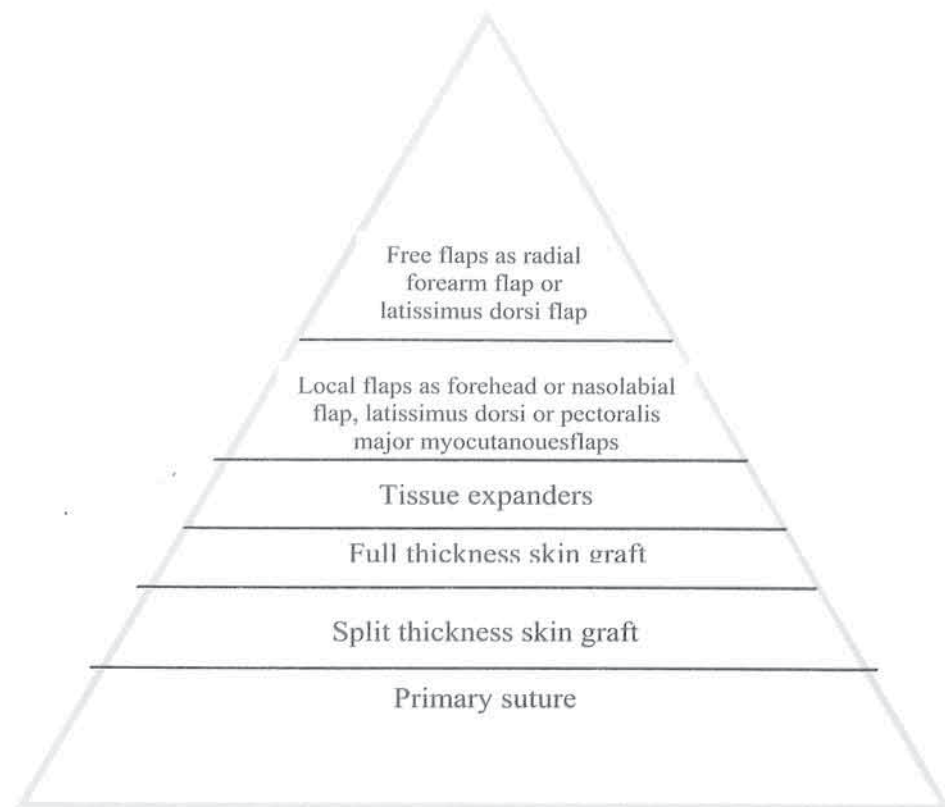


Fig. (8.21): The Reconstruction Ladder

Aesthetic surgery

By definition, this is surgery intended to improve appearance. Thus it is used for correction of any disfigurement as in scar revision and in enhancement of the normal appearance by correcting a deformed nose or correcting the effects of the aging process in the face. The selection of patients for aesthetic surgery is important, since there is no pathology to correct, but rather an improvement in the body image is asked for. Examples of aesthetic surgery are

- Rhinoplasty Correction of a deformed nose.
- Face lifting. The aim is to get rid of the redundant excess skin of the face that occurs with aging. It is done through scars that are hidden in hair or just in front of the ears in a natural crease. Recently, the procedure can be carried out through endoscopic techniques.
- Eyelids surgery. The operation is intended for correction of the aging process affecting the eyelids through very fine incisions in their creases.
- Breast surgery This is either augmentation mammoplasty to enlarge small breasts, or reduction mammoplasty. Augmentation is done by the insertion of a silicon implant under the breast.
- Liposuction. This is body sculpturing surgery by aspiration of excess fat accumulating in the abdomen, buttocks or trochanteric areas. It is done by insertion of a special cannula through a one cm skin incision. The cannula is connected to a special machine with a high vacuum pressure to allow aspiration of the excess fat. Sometimes the aspirated fat can be treated in a special manner and is reinjected to augment other areas of the body with depressions (autologous fat injection).

SURGICAL NUTRITION

Introduction

The great majority of patients who come to the service of the surgeon are fairly nourished, and return to normal physical and bowel activity shortly after the operation. A minority, however, are either malnourished at the time of presentation, or develop a postoperative problem that worsens their nutritional status. This latter group is at a higher morbidity and mortality risk, and, therefore, requires special attention. The diagnosis and treatment of malnutrition are important complementary surgical skills that improve the outcome of service.

Metabolic Considerations

The Physiological Status

Maintaining a healthy nutritional status requires the adequate and balanced supplementation of calories, proteins, vitamins, minerals, trace elements and water (Table 9.1).

- Calories are obtained either by carbohydrates, which provide 4 Kcal/g, or by fats, which provide 9 kcal/g.
- Carbohydrates are digested and then metabolized as glucose. The latter is the only source of energy for certain cells in the body, which are the brain, red blood cells, and adrenal medulla.
- Fats are accepted as source of energy by other tissues and its administration is important to provide the body with essential fatty acids. Excess calorie intake is stored as fat.
- Proteins provide the building blocks of muscles, as well as the raw material for the synthesis of enzymes and some of the hormones. Proteins are converted in the body to amino acids prior to their utilization. Essential amino acids are obtained only from dietary proteins. A practical way to assess the fate of ingested protein is to look at it in terms of its nitrogen content. The intake of 6.25 g of protein means 1g nitrogen, thus the daily requirement of nitrogen is about 0.1 g/kg. If the intake of nitrogen exceeds its amount in urine, the body is said to be in a positive nitrogen balance, i.e. an anabolic state in which proteins are being formed. If on the other hand the body is in negative nitrogen balance, then proteins are being catabolized and changed into glucose, to supply energy, by the process of gluconeogenesis. Therefore, for optimal utilization of proteins, enough calories should be supplied at a ratio of 100-250 kcal 1g nitrogen. At this ratio carbohydrates are said to have a protein-sparing effect.

CHAPTER CONTENTS

- Introduction
- Metabolic Considerations
 - The physiological status
 - The abnormal situation
- Malnutrition in surgical patients
 - Causes
 - Effects on outcome of surgery
 - Diagnosis
 - Nutritional support
- Enteral nutrition
 - Indications
 - Route of administration
 - Regimens and formulae
 - Complications
- Parenteral nutrition
 - Indications
 - Route of administration
 - Regimens and formulae
 - Monitoring
 - Complications
 - Home parenteral nutrition

Table (9.1) Recommended daily basal requirements

	/Kg
Water (ml)	35
Non-protein (Kcal)	30
Proteins (g)	0.7
Nitrogen (g)	0.1
Sodium (mmol)	1
Potassium (mmol)	1

Calories produced by 1 g of

Carbohydrates	4 kcal
Fats	9 kcal
Proteins	4 kcal

For optimal protein utilization Carbs (& fats)/Nitrogen

$$= 100-250 \text{ kcal/1g} \\ [6.25\text{g proteins} = 1\text{g Nitrogen}]$$

The Abnormal Situation

The above-mentioned fine balance may be disturbed either by starvation, or by the development of a catabolic state induced by the stress of trauma, surgery, or septic complications.

1. **The starving patient.** The body store of carbohydrate, in the form of liver glycogen is rapidly depleted within one day. Fat is then used as a source of energy. However, the noble organ, the brain is unable to utilize any source of energy other than glucose. For its sake the body is obliged to breakdown its muscle protein to produce glucose by gluconeogenesis. This catabolic process consumes 65-100 g of protein daily (250-400 g of muscle), and results in the excretion of 10-15 g of nitrogen per day. With the passage of time the brain learns to adapt to the use of some keto-acids for energy, and the nitrogen excretion falls to 3-4 g daily.
2. **The patient in catabolism.** The body reacts to the stress of trauma, and surgery by the production of excess adrenal hormones, growth hormone, and glucagon. The result is an inevitable catabolic state. Some conditions as major burns, acute pancreatitis, and major sepsis are known to raise the energy requirements to as much as 25-100% of the basal level, a situation that is termed a 'hypercatabolic state'. The provision of enough calories to such patients helps to reduce the protein breakdown, yet it cannot abolish it completely.

Malnutrition in surgical patients

Causes

The two principal causes of malnutrition in surgical wards are starvation, and the catabolism of surgery and trauma, and their complications. In many cases a combination of both exists.

Starvation

1. Social causes as poverty, and the neglected elderly.
2. Dysphagia, e.g. carcinoma of the oesophagus.
3. Loss of appetite caused by visceral malignancy, e.g. carcinoma of the stomach.
4. Repeated vomiting, e.g. pyloric stenosis.
5. Malabsorption, e.g. extensive inflammatory bowel disease, short bowel syndrome that follows extensive intestinal resection, and high output intestinal fistulae.
6. Extended postoperative restriction of oral intake due to prolonged paralytic ileus, or repeated surgical interventions.

Hypercatabolism

1. Major trauma and burns.
2. Major surgical procedures.
3. Severe acute pancreatitis.
4. Major sepsis, e.g., peritonitis and septicaemia.

Effects of malnutrition on the outcome of surgery

1. Impairment of wound healing resulting in serious consequences as burst abdomen and failure of intestinal anastomoses.
 2. Suppression of immune response and a higher susceptibility to infection.
 3. A sense of mental and physical exhaustion that delays recovery, and increases hospital stay and expenses.
 4. Reduced tolerance to radiotherapy and anticancer chemotherapy.
- In general, operative morbidity and mortality are commonly increased when some 20% of the total body weight had been lost.

Diagnosis

There is a very wide range of tests, some of which are highly sophisticated, for the assessment of the degree of nutritional impairment. Some of the simpler indications of malnutrition are mentioned below.

- Starvation and hypercatabolic states (particularly burns and sepsis) contribute to malnutrition in surgical cases.
- Malnutrition seriously affects the outcome of surgery.

Anthropometric measures

1. Recent unintentional weight loss of 10% or more.
2. Body weight is less than 80% of the ideal for height. Body mass index (BMI) = $\text{Weight kg} / (\text{Height in metres})^2$.
3. Mid-arm muscle circumference is less than 80% of value in comparable population. It correlates with the skeletal muscle mass.
4. Triceps skin fold thickness, measured by the use of a caliper, is an indication of fat loss. The average thickness in females equals 13mm and in males 11 mm.

Diagnosis of malnutrition

Anthropometric

- Recent weight loss
- BMI
- Mid-arm circumference
- Triceps skin fold

Laboratory

- Albumin
- Nitrogen balance

Immune

- Lymphocyte count
- Delayed type hypersensitivity

Laboratory tests

1. Serum albumin that is less than 35 g/L indicates a severe protein loss. A value of 25 g/L indicates a depletion of two thirds of the total body albumin.
2. Measurement of the daily nitrogen output in urine then adding to it the possible losses through other routes as intestinal fistulae, and nasogastric suction. Comparison with the nitrogen intake (grams of nitrogen = grams of protein/6.25) provides a rough idea about nitrogen balance.

Whenever the gut is functioning it should be used for providing nutrition

Immune functions

1. Total lymphocyte count below $1.2 \times 10^9/L$.
2. Impairment of delayed hypersensitivity reaction is an index of immunological affection.

Nutritional Support

Nutritional support is offered to both the malnourished, and to the patient in hypercatabolism who is likely to become so if not properly supported. The natural route of nutrition by oral intake is undoubtedly the easiest and safest, and it should be attempted whenever possible. A great deal of patience, care, and encouragement are needed for a successful oral nutritional regimen. If this is not possible, nutrition is delivered either by a tube inserted in the proximal gastrointestinal tract (enteral nutrition), or by a catheter in the venous system (parenteral nutrition).

Enteral Nutrition

The use of the gastrointestinal tract for nutritional support is generally better controlled and is less expensive than parenteral nutrition.

Indications

Enteral nutrition is indicated in patients in whom oral intake is inadequate or impossible, while having a functional accessible gastrointestinal tract.

- Unconscious patients
- Severe dysphagia

- Head and neck surgery
- Low output entero-cutaneous fistula
- Burns
- Critically ill patients who have adequate intestinal function. In any case it could either be supplemental, or total feeding.

Route of administration

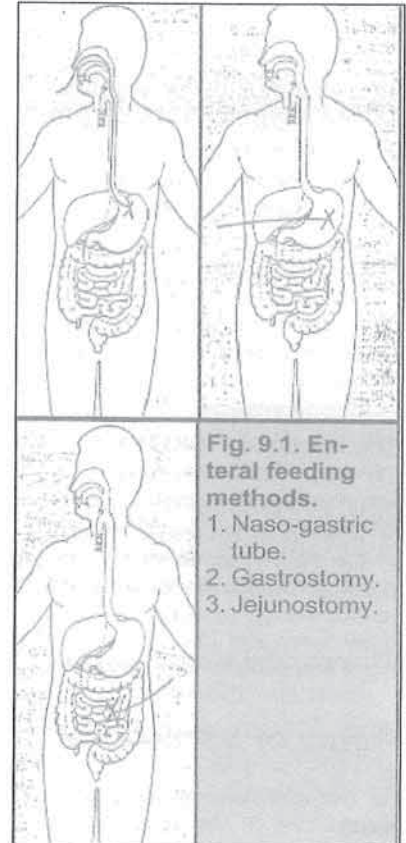
Enteral nutrition is administered through a tube inserted in proximal part of gut (Fig. 9.1).

1. Nasogastric tube
2. Gastrostomy (Fig. 9.2). Fashioning such a stoma is resorted to if nasogastric feeding is not possible because of disease or obstruction of the upper alimentary tract.
3. Jejunostomy is indicated if the stomach is diseased, or with impaired gastric emptying, and with other conditions that predispose to pulmonary aspiration as significant gastro-oesophageal reflux and loss of the gag reflex. A further advantage of a jejunostomy is that feeding can be initiated within few hours of the operation.

Administration regimens and formulae

The administration of nutrients through the stomach either by a nasogastric tube or a gastrostomy is easier to handle than the jejunostomy feeding. For the former, liquid diets formed of soups, juice, milk, and blenderized food is generally well tolerated if administered at a reasonable rate.

The jejunum, on the other hand, is highly sensitive, and feeding should be started with isotonic, sterile formulae at a slow rate, otherwise the patient develops distension, colics, and diarrhoea. The tonicity and rate are gradually increased according to the patient's tolerance. Commercially available formulas are preferred for jejunostomy feeding, these are either partially digested or elemental formulae that require no digestion and are, therefore, ready for absorption. These formulae usually supply 1000 kcal and 5 g nitrogen/L. The fat content provides 30-40% of energy, and supplies essential fatty acids. Minerals, trace elements, and vitamins are also incorporated. Two to three litres are usually adequate to cover the usual daily requirements of an adult.



Complications

The use of the gastrointestinal tract for feeding does not mean that enteral nutrition is free of complications. These are mechanical, gastrointestinal, or metabolic.

1. Mechanical complications include pharyngeal and oesophageal mucosal irritation and ulceration, obstruction of the feeding tube lumen, and tube displacement.
2. Gastrointestinal complications include nausea, vomiting and pulmonary aspiration, distention, colics, and diarrhoea which are usually related to the rapid administration of hypertonic feeds.
3. Metabolic complications include glucose intolerance, electrolyte imbalance, and nutrient excesses or deficiencies.

Parenteral Nutrition

In this form of feeding nutrients are supplied via an intravenous delivery system. When used as the sole means of providing nutrition, it is referred to as total parenteral nutrition (TPN).

Indications

Parenteral nutrition is indicated for the malnourished or the hypercatabolic patient in whom the intestine fails to absorb nutrients.

- Massive bowel resection
- Radiation enteritis
- Severe inflammatory bowel disease
- Prolonged paralytic ileus
- High output intestinal fistulae
- Moderate to severe pancreatitis

Preoperative administration of parenteral nutrition to the severely debilitated patients for a period of 10-14 days is thought to reduce postoperative mortality. On the other hand; delaying resection of a gastrointestinal carcinoma may affect long-term survival. The balance is a delicate one, and each case is judged individually.

Route of administration

As the nutritive solutions are hyperosmolar, they tend to produce thrombosis of the veins. Rapid dilution of the solutions with high blood flow is needed to reduce this effect, and it is, therefore, customary to use a central venous catheter inserted percutaneously through either the subclavian, or the internal jugular vein (Fig. 9.3), and threaded forward so that its tip will lie in the superior vena cava. Before use, the position of the catheter tip should be checked by chest X-ray. An exception is the intralipid preparation, which is isotonic and can thus be safely administered through a peripheral vein. The insertion of the catheter should be under absolute sterile conditions.

Administration regimens and formulae

Daily requirements are estimated according to the body weight, and to which are added the deficits. Daily readjustment depends upon monitoring of fluid, electrolyte and nutritional status. Carbohydrates are provided mainly by glucose. To provide enough energy without overloading the circulation, high concentrations are used, e.g., 20% or 50% glucose.

Proteins are commonly administered as L-amino acids solutions that should contain the essential members. Examples of the commercially available preparations are Vamin, and Aminosteril. These solutions also contain sodium, potassium, phosphate, magnesium, vitamins, trace elements and glucose in amounts that vary from one product to the other. For the best use of amino acids to be incorporated into the cells, 100-250 calories, that are

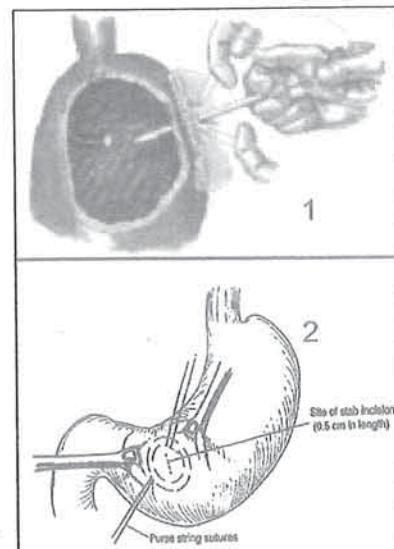


Fig. (9.2) Two types of gastrostomy

1. Percutaneous endoscopic gastrostomy (PEG).
2. Open gastrostomy.

Enteral nutrition complications

- **Mechanical**
 - Pharyngitis
 - Oesophagitis & ulceration
 - Blocked tube
 - Displaced tube
- **GIT**
 - GOR & pulmonary aspiration
 - Nausea & vomiting
 - Distension, colics & diarrhoea
- **Metabolic**
 - Hyperglycaemia
 - Electrolyte imbalance
 - Nutritional deficiency

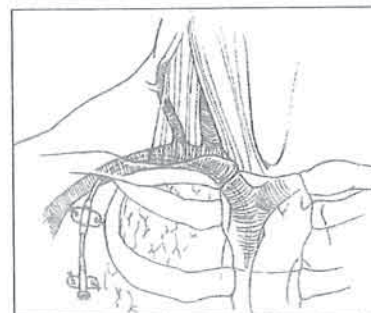


Fig. (9.3) Subclavian vein catheter

made up of glucose or fat, should be supplied for every gram of nitrogen. Amino acids and glucose are, therefore, given simultaneously, or better still both are included in a 3L-bag that is prepared under aseptic precautions in the hospital pharmacy.

Fat emulsions are produced from soya oil (Intralipid), and are available in 10% and 20% concentrations, both are isotonic and can be given via a peripheral vein. Fat emulsions are good source of energy because one litre of the 20% preparation provides 2000 kcal. They are usually given twice weekly to provide essential fatty acids. Intralipid is not to be mixed with other preparations in the same bag, and is conveniently given through a peripheral vein.

When mainly using an amino acid/carbohydrate solution, it should be administered in gradually increasing doses over 3-4 days. This allows for the development of tolerance, particularly in regard to the increased output of insulin required to deal with the carbohydrate load.

Monitoring

Monitoring is essential to determine the efficiency of parenteral nutrition, and to detect its complications at an early stage. The following are routinely performed.

1. Measuring the body weight is done daily. A weight gain of more than 300 g per day is likely to be caused by water retention.
2. Daily balance of fluid intake and output, full blood count, blood urea, serum sodium, potassium and chloride, blood and urine sugar, and nitrogen balance.
3. Thrice weekly estimation of plasma proteins, serum calcium, magnesium and phosphate, liver function tests, and blood coagulation studies.
4. Manifestations of sepsis are always searched for.

Parenteral nutrition is not physiological. The frequency and seriousness of complications require close monitoring.

Parenteral nutrition complications

- **Nutritional & metabolic**
 - Hyper or hypoglycemia
 - Na & K imbalance
 - Dehydration / overhydration
 - Deficient or excess calories
 - Hypoproteinaemia
 - Essential fatty acid deficiency
 - Vitamin deficiency
 - Trace elements deficiency
- **Catheter**
 - Misplaced catheter
 - Injury to pleura, arteries, nerves
 - Air embolism
 - Venous thrombosis & Catheter block
 - Catheter infection & septicaemia
- **Gut barrier failure**

Complications

1. Nutritional and metabolic complications arise from the administration of improper amounts of nutrients resulting in overfeeding, underfeeding, or specific nutrient imbalance as hyponatraemia, hypokalaemia, or hyperosmolar dehydration. Hyperglycaemia is a common incident that can be adjusted either by reducing the rate of glucose infusion, or by the simultaneous administration of insulin.
2. Catheter complications
 - a. Misplacement of the catheter, e.g. with a subclavian vein puncture, the catheter may be misdirected upwards towards the internal jugular vein.
 - b. Another likely error is the insertion of the catheter tip into the pleural cavity, leading to haemothorax or pneumothorax. Chest X-ray should always be done after insertion of the catheter.
 - c. Injury to arteries or to nerves of the brachial plexus is a possibility, but is rare in experienced hands.
 - d. If the infusion set is accidentally detached from the venous catheter, air can be sucked into the superior vena cava, which has a negative pressure, producing serious air embolism.
 - e. Venous thrombosis.

- f. A common serious complication is the central venous catheter infection, and septic thrombophlebitis which lead to septicaemia and, if neglected, death. The venous catheter should be introduced under strict aseptic techniques, and the entry site is inspected every other day, treated with antiseptic, and covered by sterile impermeable dressing. The drip tubing should be changed daily, and the venous line should never be used for giving or withdrawing blood or for drugs. Whenever the patient develops an unexplained fever that persists for more than 24 hours, the venous catheter should be removed, and its tip is sent for bacteriological and fungal cultures. Broad- spectrum antibiotics are commenced and are later changed according to the result of culture and sensitivity. A new central venous catheter is introduced at a fresh site.
3. Failure of gut barrier. The absence of enteral feeding leads to atrophy of intestinal mucosa. This allows for "translocation of bacteria" from the lumen to the blood stream, a situation that leads to septicaemia in the critically ill patients.

Home parenteral nutrition

Intravenous nutrition at home is usually reserved for patients who had massive small intestinal resection (short bowel). A permanent silastic central venous catheter, tunneled and cuffed is placed for long-term use. Parenteral alimentation is usually given at night over 12 hours with the aid of a mechanical pump.

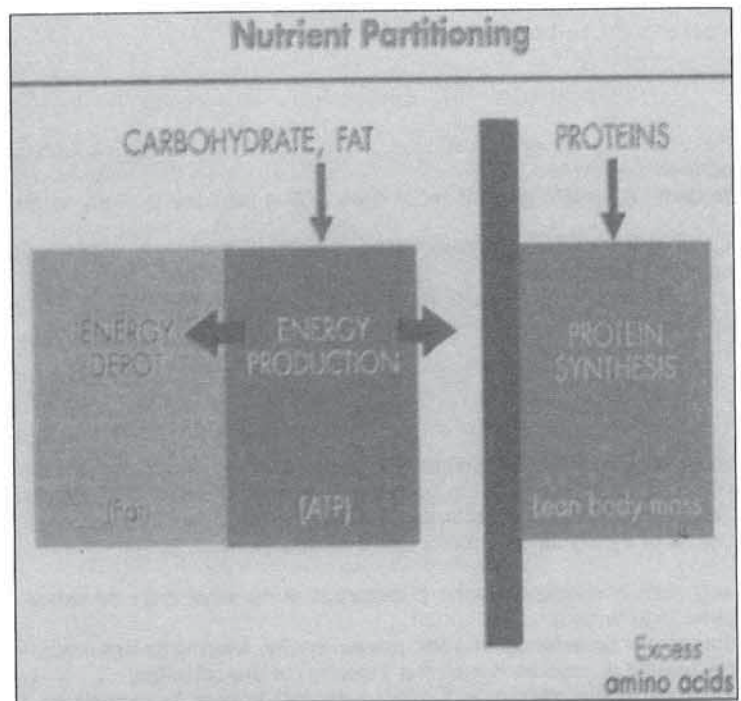


Fig. (9.4) Normal use of proteins and energy

TUMOURS

Introduction

A tumour is an autonomous, excessive, purposeless and pathological proliferation of cells. This growth continues indefinitely in the absence of physiological stimuli and without regard to its effects on the surrounding tissues.

There is a tendency for the cells of a tumour to differentiate along a particular pathway usually allowing recognition of the type of tissue cells from which it is derived. It is convenient to note that the word neoplasm is applied to all types of tumours; while the term cancer includes all types of malignant tumours. Malignant epithelial tumours are termed carcinomas and malignant connective tissue tumours sarcomas.

CHAPTER CONTENTS

- Introduction
- How cancer arises
- Aetiology of cancer
- Stages of cancer development
- Cancer grading
- Spread of cancer
- Diagnosis
- Treatment
- Prognosis
- Hope for the future

Benign tumours have the following characters

1. They are highly differentiated; the tumour cells resemble the adult cells of the tissue of origin in their morphology, in their arrangement and in their functional activities.
2. They grow slowly by expansion causing compression of the surrounding structures.
3. They are usually encapsulated. This capsule is formed essentially of the surrounding tissue.
4. They remain localized neither invading the adjacent structures nor giving rise to metastases.

The striking feature of a malignant neoplasm is its ability to invade and to metastasize. This chapter essentially deals with malignant neoplasms.

How cancer arises

Gene mutations

Malignant transformation of a cell comes about through the accumulation of mutations in specific classes of the genes within it. Two gene classes, which together constitute only a small proportion of the full genetic set, play major roles in triggering cancer. In their normal structure, they ensure normal controlled cell growth and division.

1. Proto-oncogenes encourage such growth. When mutated, proto-oncogenes can become carcinogenic oncogenes that force excessive multiplication. An example of oncogenes is erb-B which is involved in the development of glioblastoma and breast cancer.
2. Tumor suppressor genes normally inhibit abnormal cell growth and division. They contribute to cancer when they are inactivated by mutations. The resulting loss of functional suppressor proteins deprives the cell of crucial brakes that prevent inappropriate growth. Examples are
 - p53 suppressor gene. Responsible for the production of p53 protein, which can halt cell division and induce abnormal cells to kill themselves. It is involved in a wide range of cancers.
 - BRCA1 suppressor gene is involved in breast and ovarian cancers.
 - BRCA2 suppressor gene is involved in breast cancer.

Escaping the body defense mechanisms

The human body contains about 30 trillion cells. It is very likely, therefore, that thousands of them become mutated and cancerous everyday. Our bodies are equipped

with checking systems that continuously eliminate them, and only rarely does a cancer cell continue to live and to replicate producing a frank malignancy.

1. **Apoptosis.** (programmed cell death). One such system, present in each human cell, provokes the cell to commit suicide if some of its essential components are damaged. For example, injury to chromosomal DNA can trigger apoptosis. Developing cancer cells devise several means of evading apoptosis, become immortal and divide indefinitely.
2. **The immune system.** Mutated cells produce abnormal proteins. Whether in the tissues or in the blood stream, our immune system, therefore, recognizes these cells as foreign and recruits its cell-mediated and humoral mechanisms to get rid of them. For this reason an immuno-compromised person is more likely to develop a malignancy. An example is the development of Kaposi sarcoma in patients with AIDS.

Table 10.1. Histogenic classification of tumours		
Cell or tissue type	Benign	Malignant
Epithelium		
▪ Stratified squamous epith.	Papilloma	Squamous cell carcinoma Basal cell carcinoma
▪ Columnar epithelium	Adenoma	Adenocarcinoma
▪ Transitional epithelium	Papilloma	Transitional cell carcinoma
Connective tissue		
▪ Adipose	Lipoma	Liposarcoma
▪ Fibrous	Fibroma	Fibrosarcoma
▪ Cartilage	Chondroma	Chondrosarcoma
▪ Bone	Osteoma	Osteosarcoma
▪ Smooth muscle	Leiomyoma	Leiomyosarcoma
▪ Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
Neuroectoderm		
▪ Glial cells	Ganglioneuroma	Gliomas
▪ Nerve cells	Pigmented nevus (hamartoma)	Neuroblastoma & medulloblastoma
▪ Melanocytes	Meningioma	Melanoma
▪ Meninges	Shwannoma & neurofibroma	Malignant meningioma
▪ Nerve sheath		Neurofibrosarcoma
Haemopoietic and lymphoreticular tissue		Leukaemias Lymphomas
Blood vessels	Haemangioma (hamartoma) Glomangioma	Haemangiosarcoma
Lymphatic vessels	Lymphangioma (hamartoma)	Lymphangiosarcoma
Germinal and embryonic cells	Benign teratoma	Malignant teratomas Seminoma Dysgerminoma
Placenta	Hadatiform, mole	Choriocarcinoma

Aetiology of cancer

Cancer seems to arise from the effects of two different kinds of carcinogens.

1. One of these categories comprises agents that damage genes involved in controlling cell proliferation and migration. These initiate the malignant transformation (oncogenesis)
 - Chemical carcinogens.
 - Physical agents.
 - Viruses.
 - Diet.
 - Idiopathic.
2. Another category includes agents that do not damage genes but instead selectively enhance the growth of tumour cells or their precursors, e.g., hormones.

- TSH stimulates growth of papillary carcinoma of the thyroid.
- Oestrogens stimulate growth of breast cancer that contains oestrogen receptors.
- Progesterone stimulate growth of breast cancer that contains progesterone receptors.
- Androgens stimulate growth of prostate cancer

Chemical carcinogens

- Tobacco smoke is top carcinogen. Smoking, mainly of cigarettes, causes cancer of the lung, upper respiratory tract, esophagus, bladder and pancreas. Whether smoking will result in malignancy depends on several factors, including the frequency of smoking, the cigarettes tar content and-most important.the duration of the habit. The risks vary from one type of cancer to another, thus, on average, smokers are twice as likely to be afflicted with cancer of the bladder but eight times more likely to contract cancer of the lung.
- Occupational carcinogens. Workers in certain occupations are exposed to chemical carcinogens. In recent years, however, the control of such occupational carcinogens has brought about a great reduction of such malignancies.

Table 10.2. Occupational carcinogens		
Chemical	Cancer	Occupation
Asbestos	Mesothelioma of lung	Shipyard, insulation and demolition workers
Benzidine and other aromatic amines	Transitional cell carcinoma of urinary bladder	Petrochemical and rubber workers
Diesel exhaust	Bronchial carcinoma	Railroad and bus garage workers, truck operators and miners
Soot	Skin squamous cell carcinoma	Chimney sweepers

Physical agents

- Continuous mechanical irritation, e.g., by gallstones is a predisposing factor for carcinoma of the gallbladder.
- Ionizing radiation. X-ray, and alpha, beta, and gamma-rays can all induce cancer in man and animals and their effect is cumulative. They dislodge ions of water with the formation of free radicals leading to damage of the DNA. Because of its density, bone tissue absorbs ionizing radiations more than soft tissues and consequently haematopoietic tissue is severely affected. Leukaemias are among the commonest types of cancer resulting from ionizing radiations. The thyroid, breast and lung are relatively common sites. X-ray irradiation of the neck in infancy and childhood carries a significant risk of the development of thyroid cancer.
- Ultraviolet (UV) radiation. The effects of UV rays are limited to the skin as they have a poor power of penetration. Fair skinned people exposed to the sunlight for long periods have a high incidence of basal- cell and squamous-cell carcinomas, and malignant melanomas. Melanin present in the epidermis of dark skinned individuals absorbs the ultraviolet rays and protects them from skin cancer.

Viruses

Both RNA and DNA viruses have been shown to be capable of causing cancer but the commoner cancer-causing pathogens are the DNA viruses. They propagate by invading the living cells of a host and using the cells' DNA-synthesizing and protein-

making machinery to generate copies of themselves. Of these carcinogenic agents, the most important are:

- Human papilloma viruses types 16 and 18, which are sexually transmitted. They can lead to cancer of the cervix, and anus.
- Hepatitis B and C viruses, can cause hepatocellular carcinoma.
- Epstein-Barr virus, which is best known for producing mononucleosis, can produce nasopharyngeal carcinoma and Burkitts lymphoma.

Diet

- Animal (saturated) fat in general and red meat in particular are strongly linked to malignancies of the colon and rectum.
- Consumption of large quantities of alcoholic beverages, particularly by smokers, increases the risk of cancer of the upper digestive tract, and alcoholic cirrhosis frequently leads to hepatocellular carcinoma.

Idiopathic and inherited cancer syndromes

Perhaps a quarter of all cancers would develop even in a world free of external influences, simply because of the production of carcinogens within the body and the occurrence of unrepaired genetic mistakes. One long-standing theory holds that aging plays a role by increasing the generation in the body of so-called free radicals, which are chemically reactive fragments of molecules. By reacting with a gene's DNA, these fragments can damage and permanently mutate the gene. Antioxidant vitamins A, C and E are known to neutralize free radicals. Regularly eating vegetables and fruits is thought to protect against cancer. Inherited cancer syndromes are well-known e.g. familial adenomatous polyposis (FAP), hereditary non-polyposis colon cancer (HNPCC) and multiple endocrine neoplasia (MEN).

Stages of cancer development

1. **Hyperplasia.** The altered cell and its descendants continue to look normal, but they reproduce too much, a condition termed hyperplasia. After years, one in a million of these cells suffers another mutation that further loosens controls on cell growth.
2. **Metaplasia**, i.e., change of one type of epithelium into another, may occur in the way of cancer development in some cases (Fig. 10.1).
3. **Dysplasia.** In addition to proliferating excessively, the offspring of this cell appear atypical in size, shape and in orientation (relation to each other); the tissue is now said to exhibit dysplasia (Fig. 10.2).

Cells have

- i. Large distorted nuclei with high nuclear / cytoplasmic ratio.
 - ii. High mitotic activity.
 - iii. Probably multiple nucleoli.
4. **In situ cancer.** Once again, after a time, a rare mutation that alters cell behavior occurs. The affected cells

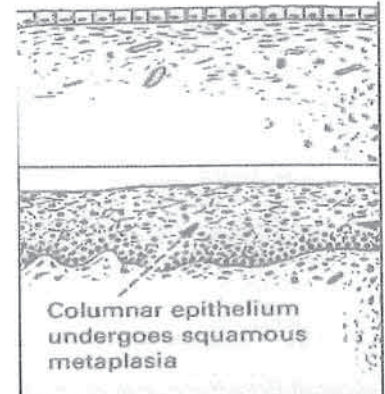


Fig. 10.1. Metaplasia.

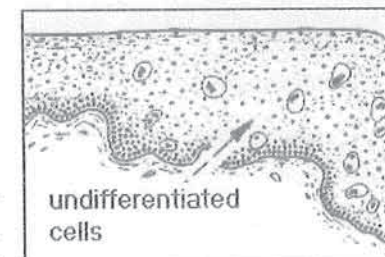


Fig. 10.2. Dysplasia.

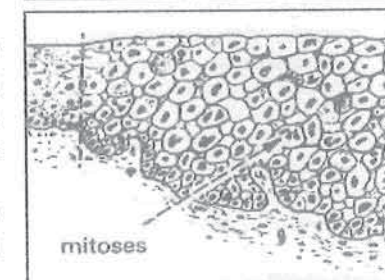


Fig. 10.3. In situ cancer. The basement membrane is intact.

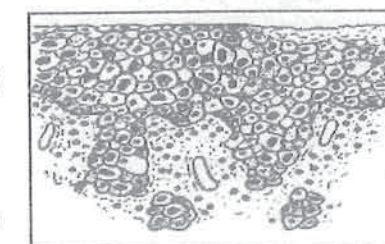


Fig. (10.4) Invasive cancer

become still more abnormal in growth and appearance. If the tumor has not yet broken through the basement membrane, it is called in situ cancer (Fig. 10.3). This tumor may remain contained indefinitely or may progress to invasion.

5. **Invasion.** If the genetic changes allow the tumor to begin invading neighboring tissue, invasive malignancy develops (Fig. 10.4). Malignant tumours grow both by expansion and by infiltrating the surrounding tissues. Growth occurs more readily along lines of least resistance such as planes of loose connective tissue. Fibrous fascia, cartilage and bones are more resistant. The rapidly growing malignant tissue stimulates the formation of new capillaries from adjacent blood vessels in order to sustain its nutrition and oxygenation; a process that is called 'tumour angiogenesis'. Frequently, however, the tumour outgrows its blood supply leading to ischaemic death of the cells especially those that lie farthest from the blood supply. Clinically this manifests as central necrosis.
6. **Metastasis.** Many invasive cancer tend to shed cells into blood or lymph to establish metastases throughout the body.

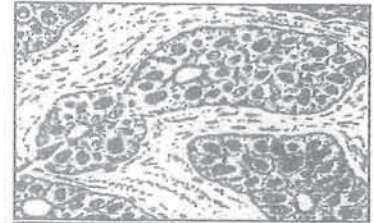


Fig. 10.5. Well-differentiated adenocarcinoma. Attempts at formation of acini.

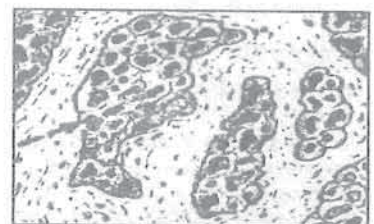


Fig. 10.6. Undifferentiated adenocarcinoma.

Cancer grading

Grading is a measure of tumour aggression. This largely depends upon its differentiation.

- Well-differentiated tumours have a high content of cells that resemble the tissue of origin (Fig. 10.5). These are the least aggressive.
- Moderately-differentiated tumours.
- Poorly-differentiated and undifferentiated tumours bear little or no resemblance to the tissue of origin (Fig. 10.6). These are the most aggressive as they grow, divide, invade and spread rapidly.
- The rate of cellular production depends on the number of cells undergoing mitosis and on the time they take to complete the mitotic cycle. The mitotic index means the proportion of cells seen to be in mitosis in a histological section of the tumour.

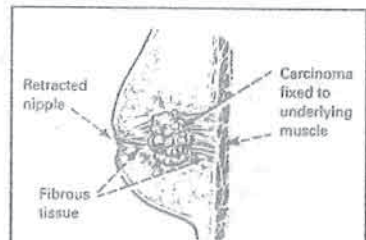


Fig. 10.7. Local spread.

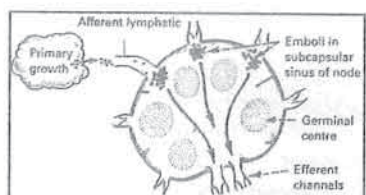


Fig. 10.8. Lymphatic spread.

Spread of Cancer

Properties that allow metastasis

Acquiring the capabilities needed to emigrate to another tissue is a key event in the development of cancer. To metastasize successfully, cancer cells have to detach from their original location, invade a blood or lymphatic vessel, travel in the circulation to a distant site and establish a new cellular colony. At every one of these steps, they must escape many controls that, in effect, keep normal cells in place.

Cancer cells have the following abilities that allow metastases

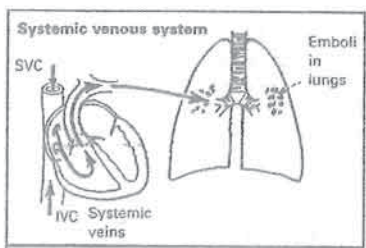


Fig. (10.9) Blood-born spread to the lungs

1. Defective cell adhesion. Malignant cells lack the adhesion proteins that bind the cells to each other forming tissues. They are, therefore free to detach and migrate.
2. Tumour angiogenesis allows proximity of malignant cells to the newly formed capillaries and so enhances easy access to the circulation.
3. Production of proteolytic enzymes that digest the basement membrane allowing invasion, and digest capillary and lymphatic wall to allow them access to the lumen.
4. They escape apoptosis when they reach some tissue different from their origin.

Modes of metastasis

1. **Local spread**, first into the tissues of the same organ, then into neighbouring organs (Fig. 10.7).
2. **Lymphatic spread**. Malignant cells very frequently invade the endothelium of the lymphatic capillaries and then either grow inside the lymphatic vessels (permeation) or are carried as emboli to the draining lymph nodes (embolization). The cells may then be carried by the efferent lymphatics to the next group of lymph nodes (Fig. 10.8). Obstruction of lymph nodes may lead to oedema in the drainage area and also in reversal of lymph flow.
3. **Blood-born spread**. Malignant cells can also invade the walls of the thin capillaries, and so tumour emboli are carried to distant organs leading to micrometastases which may remain silent for a long time or grow leading to gross distant deposits, in the lung, liver, bones, and brain. It should be stressed that not all the malignant cells which gain access to the circulation are going to produce metastases. This tumor angiogenesis is due to the secretion of certain growth factors secreted by tumours cells as fibroblast growth factors and vascular endothelial growth factors.

Fortunately, even when cancer cells do get into the circulation, the formation of secondary tumours is not inevitable. Probably fewer than one in 10,000 of the cancer cells that reach the circulation survive to establish a new tumour at a distant site. Blood circulation explains much about why various metastatic cancers spread preferentially to certain tissues. Circulating tumor cells usually get trapped in the first vascular bed (or network of capillaries) that they encountered "downstream" of their origin. The first vascular bed encountered by blood leaving most organs is in the lungs (Fig. 10.9) only the gastrointestinal tract sends its blood to the liver first through the portal vein (1010). Accordingly, the lungs are the most common site of metastasis, followed by the liver. The latter is the commonest site of deposits from the gut.

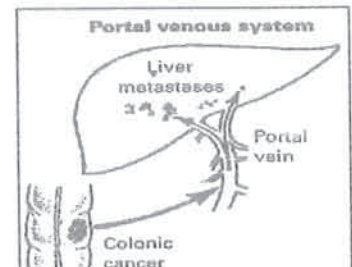


Fig. 10.10. The liver is the first organ to be affected by Metastases from GIT malignancies.

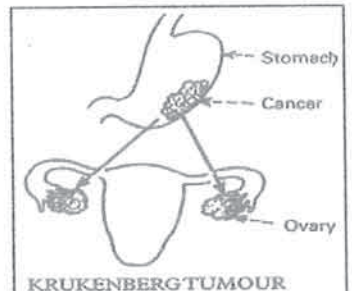


Fig. 10.11. Trans-coelomic spread.

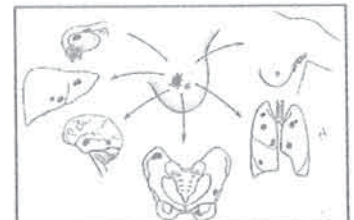


Fig. 10.12. Explosive concept of cancer dissemination.

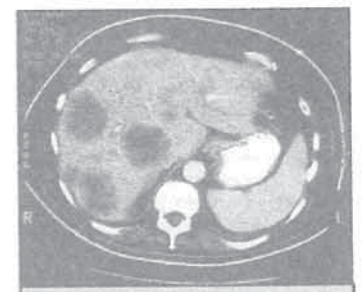


Fig. (10.13) Multiple liver metastases detected on CT scan

4. **Trans-coelomic spread.** Malignant cells travel along cavities. An example is the spread of carcinoma of the stomach down to the ovaries (Fig. 10.11).

Stepwise versus explosion concept of metastasis

Formerly it was thought that malignant dissemination takes place at two steps, the first is lymphatic, while the second is blood-born. This concept is no longer acceptable, and we now know that even at the earliest stage tumour cells invade both the lymphatics and the blood vessels simultaneously. In other words as the tumour spreads it actually explodes in all directions (Fig. 10.12). Regional lymph node involvement is no longer regarded as a stage in the progression of the disease but as an indicator that widespread dissemination has occurred. Table (10.3) illustrates the main biological features of cancer cells.

Table (10.3): The main biological features of cancer cells

Feature	Main mechanism
1. Autonomous growth	Normal cells grow within a well-defined cell cycle. The cycle is much more rapidly activated by certain molecules; cyclins which enhance growth and inhibit the checkpoints of the cycle.
2. Ignoring anti-proliferative signals	Ignoring the signals of tumour suppressor genes as p53 or transforming growth factor beta (TGF β).
3. Invasion and dissemination	Loss of cellular adhesion molecules as cadherins which bind the cells together and loss of integrins which bind the cells to the surrounding intercellular matrix.
4. Aversion to apoptosis	Mutation of the Bcl-2 gene family which mediates apoptosis.
5. Increased angiogenesis	Increased expression of the vascular endothelial growth factor (VEGF).

Staging

Staging is an expression of the extent of malignancy and, hence, reflects the likely prognosis. The most widely used staging system is the TNM in which the three components of the disease are assessed. These are

- Extent of primary tumour (T), i.e., its size, depth of invasion, and invasion of nearby organs.
- Presence or absence, and extent of lymph node involvement (N).
- Presence or absence of distant metastases (M).

Diagnosis

Early detection of asymptomatic cases (screening)

Some people may have a higher risk of developing a certain malignant tumour. In this case it is sensible, to follow certain screening programs to detect the neoplasm as early as possible. Common examples are

1. Annual soft tissue mammography for females who have a higher chance of developing breast cancer. These include patients who already had carcinoma of the breast on one side, the first-degree relatives of patients with breast carcinoma, or patients who had excision of benign dysplastic lesions.
2. Annual colonoscopy for patients with ulcerative colitis who have a high risk of developing cob-rectal cancer. These are patients with pancolitis of more than ten year duration.
3. Annual prostate specific antigen (PSA) for men above fifty.

Diagnosis of symptomatic cases

The aim is to diagnose the disease and to determine its extent (stage). In addition to thorough clinical assessment, the following diagnostic aids are commonly used.

1. **Radiological.** Various radiological techniques including contrast studies, ultrasound, CT (Fig. 10.13) and MRI are utilized for diagnosis.
2. **Endoscopy.** This is very useful for diagnosis of most lesions of the respiratory, gastrointestinal and urinary systems. High resolution endoscopic ultrasound (EUS) incorporates the U/S probes into the endoscope. Excellent cut sections displaying the depth of tumours can be seen e.g. cancers of the esophagus, stomach, pancreas or rectum.
3. **Tumour sampling**
 - Tissue biopsy; needle, endoscopic or operative biopsies obtain a piece of tissue from which sections are made for histological examination. Diagnosis of malignancy depends on both cellular morphology as well as orientation and invasion. The latter is the specific feature of malignancy. Histopathological findings are the most dependable.
 - Cytology The loss of cellular adhesion that is characteristic of malignancy allows easy retrieval of cells either by a needle aspiration or by a brush from surface lesions. Fine needle aspiration cytology is now a well-established line of investigation, which is commonly used to diagnose lesions of the thyroid, breast, liver or prostate. Ultrasound or CT-guided needle biopsy is performed for retroperitoneal or renal lesions. Diagnosis depends on cellular morphology only, as there only cells and not tissues under the microscope. The test, therefore, requires an expert cytologist.
4. **Tumours markers**
 - Many malignant tumours secrete certain oncofetal proteins which can be estimated. This may help in the diagnosis of certain tumours and in the follow-up of the patients after tumour excision. Examples include
 - Alpha-feto protein is raised in hepatocellular carcinoma and in non-seminomatous testicular tumours.
 - Carcinoembryonic antigen is raised in carcinoma of the gastrointestinal tract, pancreas and breast.
 - Prostate specific antigen is raised in prostatic carcinoma.
 - CA 15-3 is raised in carcinoma of the breast.
 - CA-125 is raised in carcinoma of the ovaries.
 - CA 19-9 is raised in carcinoma of the gastrointestinal tract and pancreas.
 - Thyroglobulin is raised in carcinoma of thyroid.
 - Tumours arising from the endocrine glands, especially if benign, secrete the hormones of the glands from which they arise. Examples include
 - Pituitary tumours secrete trophic hormones as ACTH.
 - Pancreatic islet tumours secrete insulin, glucagon or somatostatin.
 - Parathyroid tumours secrete parathormone.
5. **Isotopic scanning** As positron emission tomography (PET) utilizes a fluorine tagged tracer as flouro-desoxy-glucose for a functional scan detecting tumours with high glucose metabolism. It is sensitive for the detection of small metastases. Anatomical sites of such metastases can be more accurately determined if combined with a CT scan (PET-CT). Together with laparoscopy, both are the only two means available so far for the detection of peritoneal nodules or small liver metastases.

Treatment

Prophylactic treatment

Certain precancerous lesions are well known and their eradication can prevent the development of cancer. A well known example is familial polyposis of the colon which is one hundred per cent premalignant. Total proctocolectomy protects the patient from the subsequent development of large bowel cancer.

Treatment of established cases

There are many modalities for treatment of a patient with cancer and the best chance for the patient will be offered when a multidisciplinary approach is practiced. The patient should be examined by a team of a surgeon, radiotherapist and medical oncologist to decide the best plan for management.

After thorough clinical assessment and investigations the tumour is staged. Broadly speaking, it lies under one of two categories

1. Early (equivalent terms = loco-regional, potentially curable, operable) cancer.
 - There is no evidence of distant spread.
 - Cure is possible if this local disease is eradicated.
 - Treatment is radical, i.e., aims at cure.
 - Treatment is, essentially, by loco-regional modalities, i.e., surgery, radiotherapy, or by a combination of both.
 - Adjuvant (complementary) treatment of systemic modalities such as chemotherapy is indicated if there is a high possibility of systemic microscopic spread in distant sites. This is usually expected if the tumour reaches the lymph nodes. The larger the number of involved nodes, the higher is the possibility of microscopic metastases.
2. Late (equivalent terms = systemic, incurable, inoperable) cancer.
 - There are distant metastases.
 - Cure is not possible.
 - Treatment aims at palliation of the patient's symptoms so as to provide him with a reasonable life quality.
 - As the disease is systemic, treatment is also essentially by systemic modalities as chemotherapy and hormones.
 - Surgery or radiotherapy is sometimes needed to palliate local symptoms. In the gastrointestinal tract the best palliation is achieved by surgical excision. Cancer pain is controlled either by analgesics or by neurosurgical operations that interrupt the pain pathway.
 - Pain relief is commonly accomplished by the administration of non-steroidal anti-inflammatory agents or narcotic analgesics. In severe cases neurosurgical procedures to interrupt pain pathway may be needed.

The individual modalities of treatment include

1. Surgery

- Primary tumour. Radical surgery aims at excision of the primary tumour with as wide a safety margin so as to ensure removal of its microscopic extensions.
- Lymph nodes. The treatment of lymph nodes varies from one tumour to another. In gastrointestinal tract malignancies lymph nodes are routinely resected, for breast cases they are either resected or irradiated, while in head and neck and in skin cancers the nodes are treated only if they prove to contain malignant deposits. Whenever possible lymph nodes are removed in continuity with the primary tumour, the so-called block excision.

- Precautions. During surgery care is taken to avoid spillage of malignant cells. In some sites, e.g., colon and testis it is usual to ligate the main vessels before the tumour is mobilized so that shedding of cells in the circulation is avoided.
- Advantages. Surgical excision is both quick and effective. It accounts for the largest number of cures. Surgery is also the one method of therapy that offers the opportunity to confirm that a tumour has been fully excised, because a pathologist can examine the specimen removed to ensure a clear safety margin.
- Disadvantages and limitations. Surgery may produce functional and cosmetic disabilities. In addition, it cannot be applied if the tumour is fixed to a vital structure or if it has produced distant spread.

2. Radiotherapy

- May replace surgery or may be given in addition.
- Common indications. Cancer of the larynx so as to preserve the voice, early Hodgkin's disease, early prostate cancer, and as a part of conservative therapy for early breast cancer (after surgery).
- Method. Powerful X-rays, gamma rays, electrons, or heavy particles are directed to the tumour by one of two main methods. The radiation may be aimed at a tumor from outside the body (teletherapy), or it may be delivered by placing radioactive pellets or needles at the cancerous site (brachytherapy).
- Advantages
 - Because healthy tissues can recover from radiation exposure more readily than cancerous cells, radiation therapy can preserve the anatomical structures that surround a cancer growth, thus curing the cancer without sacrificing the patient's ability to function.
 - Radiation can destroy microscopic extensions of cancerous tissue around a tumour that a scalpel might miss.
 - Radiation is a safer option for older, frailer patients who might have difficulty recovering from surgery.
 - Patients treated with radiation usually do not require hospitalization.
- Disadvantages
 - While some tumours as squamous cell carcinoma are sensitive to irradiation, adenocarcinoma, in general, is much less sensitive.
 - Radiation is commonly associated with burns of the skin or enteritis, which are difficult to treat.
 - Compared to surgery, radiotherapy is slower as it usually takes five to eight weeks. On the other hand, just like surgery, it is not suitable for metastatic cases because irradiation of the whole body carries the risk of bone marrow depression.

3. Chemotherapy

- Common indications. Chemotherapy is the main line of treatment of leukaemias. For solid tumours these drugs are used either as the main modality in case of detected metastases, or as an adjuvant to surgery in early cases where microscopic metastases are possibly present.
- Better results are obtained from combination chemotherapy rather than using one agent. Chemotherapeutic drugs typically operate on human cells much as do some antibiotic on bacteria; they prevent cells from multiplying by interfering with their ability to replicate DNA.

- **Advantages.** The drugs travel through the circulation and can reach malignant cells anywhere in the body. Many malignancies including leukaemias, lymphomas and testicular cancer are now successfully treated by new combination of chemotherapy.
- **Disadvantages.** The available chemotherapeutic drugs often kill many healthy cells and thus bring on serious side effects. They have a tendency to damage to the rapidly growing cells of the bone marrow, for instance, causes anemia, leucopenia and thrombocytopenia. Other side effects of chemotherapy include diarrhoea, nausea vomiting and hair loss.

In order to decrease the systemic effects, the chemotherapy may be targeted into the tumour using transarterial catheters e.g. transarterial chemoembolization (TACE) for hepatocellular carcinoma.

- **Neoadjuvant therapy** means giving the chemotherapy before surgery to decrease the tumour burden aiming to downstage the tumour allowing better results of radical surgery.
4. **Hormone therapy** Hormone-blocking and hormone-supplementing therapies affect the rate at which tumor cells grow, and multiply. Examples are
 - Antioestrogens for women with breast cancer that is positive for oestrogen receptors.
 - Androgen blockade for men with prostate cancer.
 - Thyroxin to suppress TSH for patients with papillary carcinoma of thyroid. Hormone therapy has relatively mild side effects, because its actions are limited largely to tissues with receptors for specific hormones.
 5. **Immunotherapy**
 - **Non-specific.** The tuberculosis vaccine BCG stimulates the immune system in general, and was found to induce remission in cases of transitional cell carcinoma of the urinary bladder.
 - **Specific.** Administration of monoclonal antibodies from a single clone of lymphocytes that have been stimulated by a specific protein of the cancer cells. This method is still of limited use. It is currently in the stage of refinement and holds a great promise for the future.
 6. **Biological therapy** Tremendous advances in oncology and molecular biology allowed the emergence of newer anti-cancer drugs which antagonize certain biological agents expressed by certain tumours as the next examples.

Drug	The biological agent antagonized
Herceptin (trastuzumab) used mainly for breast cancer	Her-2 neu receptors
Gleevac (imatinib), used mainly in gastro-intestinal stromal tumours, GIST	Tyrosine kinase

Prognosis

Prognosis of malignancy is expressed in terms of survival, e.g., 5 , or 10-year survival rate than in terms of cure. This is because we are never sure that all microscopic disease has been eradicated. Practically speaking, however, a normal duration of life without further clinical evidence of disease is generally accepted as evidence of cure even though microscopic deposits of tumour may still be present.

Factors affecting prognosis**1. Tumour**

- Stage is the most important factor. The size, depth of invasion, lymph node involvement, and the presence of distant deposits all affect the prognosis.
- Grade of malignancy and differentiation.
- Site. An example is the better prognosis of carcinoma of the distal stomach than that of the cardia.
- Type. Basal cell carcinoma, squamous cell carcinoma, and melanoma are three malignancies that arise from the skin but vary greatly in prognosis.
- The finding of reactive hyperplasia on histological examination of lymph nodes is thought to be an indication of good immunity and, hence, a better prognosis.

2. Management

- Early detection in the pre-symptomatic phase greatly improves the outcome of treatment.
- Prompt treatment in a specialized centre improves prognosis.

3. Patient

- Age of patient.
- Presence of concurrent debilitating illness.
- Immuno-suppression.

In fact many malignant tumours are curable, e.g., most of skin cancers, leukaemias and lymphomas. In other situations the outcome is not that successful and until now malignancy still represents a nightmare

Hope for the future

1. Wider application of early detection programs.
2. Refinement of surgical and radiotherapy techniques.
3. Production of more effective chemotherapy with less side effects.
4. Development of immunotherapy.
5. Introduction of newer modalities of treatment
 - Gene therapy. Finding out the defective genes and repairing them before the production of neoplasia.
 - Introduction of drugs that fight tumour angiogenesis.

Points to Remember

- Viruses, radiation and carcinogens can lead to DNA mutation which cause uncontrolled cell growth ultimately proceeding to cancer (Fig. 10.14).

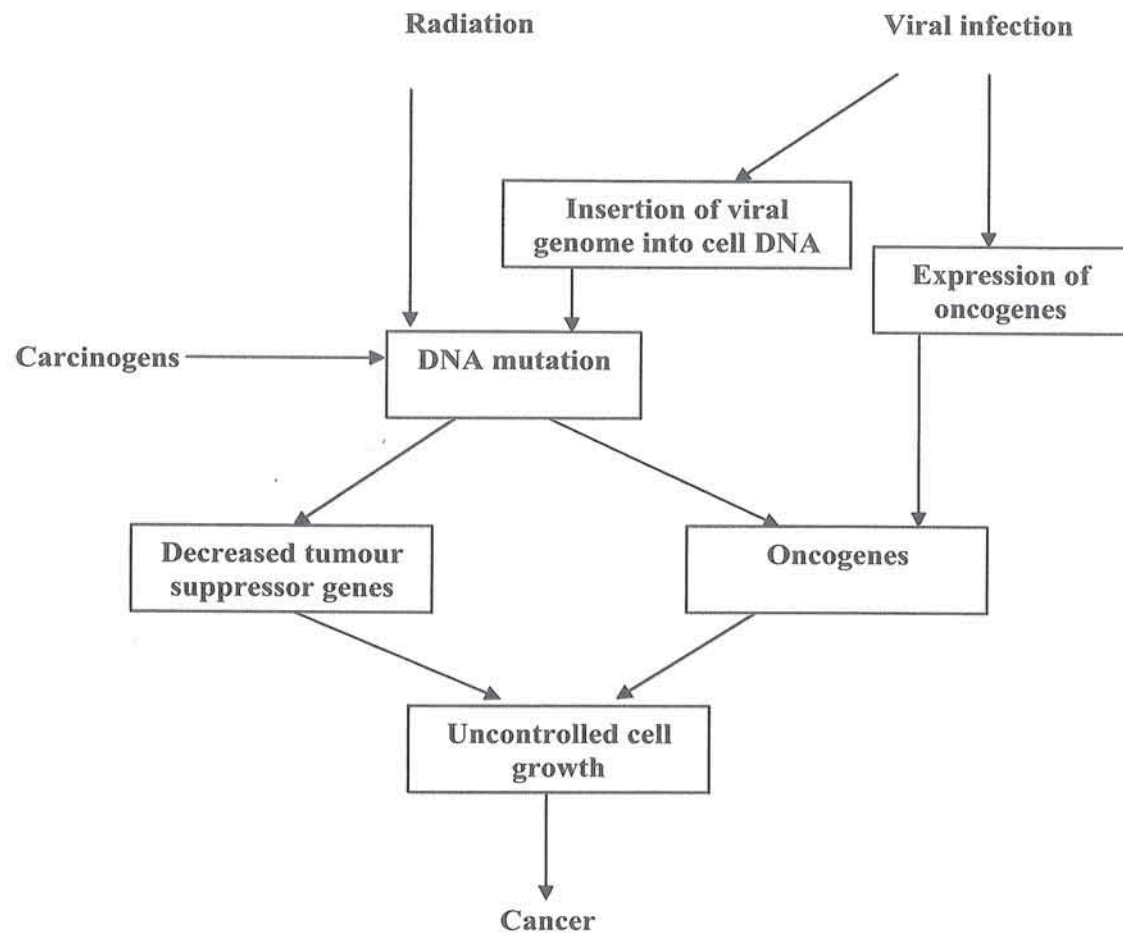


Fig. (10.14): The mechanisms of carcinogenesis

ORGAN TRANSPLANTATION

Introduction

Transplantation started early with skin, corneal and bone transplants. Whole organ transplantation started in the second half of the twentieth century, and it is now firmly established as the major form of treatment for patients with end-stage failure of the kidneys, liver, heart and heart-lung. Pancreatic and bone-marrow transplantation are achieving increasing success rates. Rapid progress in clinical transplantation is the result of an expanding knowledge of the immunological mechanisms of rejection and the ability to manipulate the immune response by the use of powerful immunosuppressive drugs.

CHAPTER CONTENTS

- Introduction
- Transplantation immunology
- Organ donation
- Renal transplantation
- Liver transplantation
- Pancreatic transplantation
- Heart transplantation
- Bone marrow transplantation

Transplantation immunology Human Major Histocompatibility Complex

Glycoprotein molecules on the surface of all somatic cells act as 'self-markers' which are responsible for triggering the immune reaction leading to allograft rejection. These molecules were originally detected on leucocytes and, are, therefore, named Human Leucocyte Antigens (now universally referred to as HLA antigens).

These HLA 'antigens' are genetically controlled by loci on the short arm of chromosome 6. This area on chromosome 6 is termed the Major Histocompatibility Complex.

The important antigens in transplantation are the Class I HLA; A, B and C antigens and the class II products HLA; D antigens. Foreign class I and class II antigens are capable of stimulating the recipient immune mechanism and triggering lymphocytes sensitization, starting rejection of the transplanted organ.

Definitions

- **Autografts:** Same individual is donor and recipient.
- **Isograft:** Tissue transfer from an identical twin, No rejection.
- **Allograft:** Donor and recipient are genetically dissimilar but of the same species.
- **Xenograft:** Donor and recipient belong to different species (animal to man). Not practical.

Immune-compatibility testing

The aim is to assess the compatibility of the recipient and the donor before undergoing organ transplantation

1. ABO blood grouping and cross-matching. This is essential for all allo-transplants. The aim is to prevent hyperacute rejection.
2. HLA cross-matching. This is essential for renal and bone marrow transplantation, and is also important for pancreas transplantation. Donor's lymphocytes are mixed with the recipient's serum and a complement. Histocompatibility provides a better chance of long-term graft survival. The test, however, is not practical to do and does not affect the prognosis of neither liver nor heart transplantation.

The rejection process

The events leading to allograft rejection involve numerous facets of the immune system but may be summarized as follows (Table 11.1):

1. Cellular mechanism

- a. The graft alloantigens are shed in a cellular or soluble form. They are engulfed and processed by the recipients macrophages, which present them to the lymphocytes.
 - b. The initial recipients response is activation and proliferation of T-helper cells.
 - c. T-helper cells produce interleukin 2 (IL-2) which stimulates the activation and proliferation of cytotoxic T-lymphocytes.
 - d. Stimulated cytotoxic T-lymphocytes attack the transplanted tissues directly and cause damage to the cells carrying the foreign HLA antigens.
2. Humoral mechanism The presence of complement-fixing cytotoxic antibodies, prior to transplantation, produces hyperacute allograft rejection by attacking vascular endothelium in the transplanted organ leading to platelet aggregation and thrombosis.

Table (11.1) Types of graft rejection

Type	Onset	Mediator	Treatment	Prognosis
Hyperacute	Immediate	Humoral ABO	Graft removal	Graft loss
Accelerated	2-5 days	Humoral or cellular	Graft removal	Graft loss
Acute	First month	Cellular	High dose steroid, OKT3	Good
Chronic	Several months	Cellular and/or humoral	None (irreversible) or retransplantation	Bad

Immunosuppressive therapy

Immunosuppressive therapy is given to all transplant recipients in order to inhibit the rejection process. The three major drugs are corticosteroids (prednisolone), azathioprine (imuran), and cyclosporine. According to their mode of action drugs used in clinical immunosuppression are classified into 2 groups

1. Drugs which deplete circulating lymphocytes Corticosteroids, antilymphocytic globulin (ALG), antithymocyte globulin (ATG) and monoclonal antibodies (OKT3).
2. Drugs which inhibit lymphocyte activation or proliferation Cyclosporine, tacrolimus (FK-506), azathioprine, mycophenolate mofetil (cellcept) and rapamycin (sirolimus).

Table 11.2 Immunosuppressive drugs

Name	Mode of action	Specific complications
Steroids	Reduce circulating lymphocytes Inhibit IL formation	DM, hypertension, weight wasting, osteoporosis and peptic ulceration
ALG & ATG	Clearance of lymphocytes through complement mediated lysis	Leucopenia Thrombocytopenia
OKT3	Monoclonal antibodies specific against T-cell receptors	Allergic and inflammatory reactions
Cyclosporine	T-cell suppression Blocks release of IL-2	Nephrotoxicity
Tarcolimus (FK506)	Similar to cyclosporine but is much more potent.	Nephrotoxicity
Azathioprine (Imuran)	Inhibits division and differentiation of lymphocytes	Hepatotoxicity Leucopenia
Mycophenolate mofetil, cellcept	Prevents lymphocyte proliferation	Leucopenia and thrombocytopenia
Rapamycin (sirolimus)	Prevents IL-2 cellular proliferation	Dyslipidaemia

Methods of Immunosuppression

1. **Induction immunosuppression.** The first two weeks after transplantation are very important in preventing the lymphocytes from attacking the transplanted organ. This is achieved by
 - a. Large doses of corticosteroids
 - b. Azathioprine
 - c. ATG.

2. Maintenance immunosuppression

- a. Cyclosporine
- b. Small doses of corticosteroids
- c. Azathioprine.

3. Anti-rejection treatment

- a. OKT3 is a monoclonal antibody against T -lymphocytes carrying CD3 receptors.
- b. High doses corticosteroids.

Complications of immunosuppression

In addition to the specific complications (table 11.2) of each drug all immunosuppressive drugs have two more common complications.

1. Infections. Various viral, bacterial or fungal infections may arise in the respiratory or urinary tracts, intra abdominally or at cannulation sites. Cytomegalovirus (CMV) infection is particularly common.
2. Neoplasia. Prolonged immunosuppression predisposes to the development of skin cancer, lymphomas and cervical carcinoma.

Organ donation**Donor types**

The organs may be taken from:

1. **Living donor** This is done only for paired organs like the kidney. It can be also used for vascularized segmental pancreatic transplantation. Segmental transplantation of the liver from a living donor is now being performed. A living related donor offers better results than a non-related volunteer. The donor should enjoy a good health and should be examined, investigated, tissue typed and cross-matched with the recipient.
2. **Cadaver donors** This method avoids the risk of complications, which may occur to the living donors and has the advantage that multiple organs can be taken from one cadaver and transplanted to many patients. The organ is removed from a patient with proved brain death. Victims of intracranial haemorrhage or head injury are the main source of donor organs.

Diagnosis of brain stem death In addition to the flat EEG, the following should be checked

- a. The patient must be in apnoeic coma; unresponsive and dependent on mechanical ventilation.
- b. No pupillary response to light, direct or consensual.
- c. No corneal reflex.
- d. Absent gag reflex
- e. Absent cough reflex via bronchial stimulation by a catheter in the endotracheal tube.
- f. Absent vestibule-ocular reflex; no eye movement upon injection of 50 ml of ice-cold water into each external auditory meatus.

Exclusions

- Drug effects as sedatives, hypnotics or muscle relaxants.
- Hypothermia, core temperature must be above 35 degrees. Low body temperature can induce deep coma.
- Metabolic abnormalities e.g. hypo or hyperglycaemia, hypo or hypernatraemia, uraemia or hepatic encephalopathy.
- Intoxication by alcohol or other drugs.

All the previous tests should be repeated after 24 hours and should be done by a separate physician, not involved in the transplant procedure. The patient's heart will

continue beating to perfuse the tissues by the use of adequate artificial ventilation, fluids, plasma, vasopressors and diuretics, until all needed organs are surgically removed,

Organ Preservation

Organs from cadaver donors can be preserved until transplanted by

1. Cooling (surface or perfusion) down to 0-4°C to reduce tissues metabolism
2. Perfusion of a special fluid medium to prevent cellular oedema and maintain vitality, e.g., University of Wisconsin (UW) solution.

Renal transplantation

Kidney transplantation is the longest established and most successful of organ transplantations.

Indications

All cases of "end-stage renal disease", which may be due to

1. Chronic glomerulonephritis.
2. Hypertension
3. Chronic pyelonephritis
4. Lupus nephritis
5. Diabetes
6. Polycystic disease
7. Obstructive uropathy

If a related living donor is available and willing, then early transplantation is indicated even before the start of haemodialysis. If the living related donor is not available, the patient is immediately placed on a cadaver donor list.

Technique (Fig. 11.1)

- The grafted kidney is placed in an extraperitoneal position in the iliac fossa (preferably the right one).
- The arterial anastomosis is performed between the renal artery and the external iliac artery (end to side anastomosis), or between the renal artery and internal iliac artery (end to end anastomosis).
- The venous anastomosis is performed between the renal vein and the external iliac vein (end to side anastomosis).
- The ureter of the graft is anastomosed to the patient's urinary bladder using the submucosal tunnel technique as an antireflux procedure.

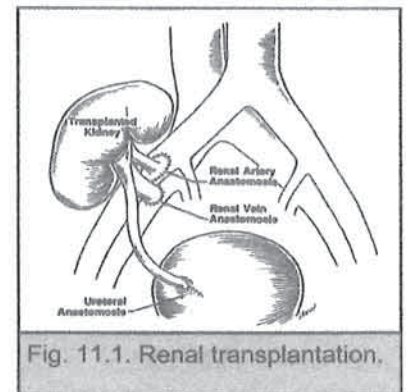


Fig. 11.1. Renal transplantation.

Complications

- Technical complications include arterial and venous occlusion, vascular stenosis, urinary leakage, ureteric stricture, wound infection and dehiscence.
- Rejection. The condition is suspected if urine output diminishes and serum creatinine rises. Percutaneous biopsy confirms the diagnosis.
- Complications of immunosuppression.
- Recurrence of the original disease in the grafted kidney.

Results

Cadaver donor	95% graft survival at 1 year 60% graft survival at 5 years
Living donor	98% graft survival at 1 year 90% graft survival at 5 years

Liver transplantation

Indications

"End-stage liver failure" due to

1. Various types of cirrhosis (alcoholic, post-viral, biliary, haemochromatosis, Wilson's disease).
2. Biliary atresia is the leading indication in children.
3. Chronic viral hepatitis.
4. Fulminant acute hepatitis.
5. Sclerosing cholangitis.
6. Budd Chiari syndrome.
7. Advanced primary liver malignancies.

Technique

(A) Cadaveric liver-transplantation;

- The whole liver of the cadaver is removed (donor hepatectomy), including the segment of inferior vena cava (IVC) embedded in the liver.
- The recipient liver is removed (recipient hepatectomy).
- The new liver graft is placed in the same position of the original liver (orthotopic transplantation) and the following anastomoses are performed in order
 - Suprahepatic followed by infrahepatic IVC anastomosis.
 - Portal vein of recipient to donor.
 - Hepatic artery of recipient to donor.
 - Common bile duct of recipient to donor.

Alternatively cadaveric liver is split into two parts ex-vivo which are transplanted into two recipients (split liver transplantation).

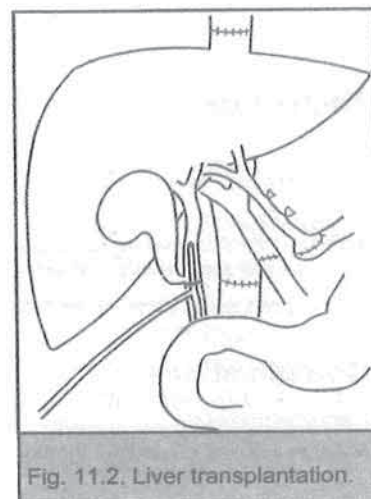


Fig. 11.2. Liver transplantation.

(B) Living donor liver transplantation

- The left lateral segment is transplanted in children whereas the right or left lobes are transplanted in adults after removal of the recipient liver.
- Donor safety is extremely important. Detailed anatomical studies of the biliary tract by MRCP and the liver vasculature by triphasic CT are essential.
- The recipient should have a liver graft weighing 1% of his body weight.
- The graft size can be estimated preoperatively using donor CT volumetry.

Complications

- Technical complications include arterial and venous occlusion or stenosis, bleeding and bile duct leakage and/or stenosis.
- Rejection.
- Recurrence of the original disease in the grafted liver.
- Complications of immunosuppression

Results

- 1 year graft survival is 85%
- 5 years graft survival is 50%

Pancreatic transplantation

Indications

1. Type I insulin dependent diabetes mellitus with renal failure. Combined renal and pancreatic transplantation is performed.
2. Patients with previous renal transplantation suffering from severe diabetes endangering the grafted kidney.

Technique

There are two types of pancreatic transplantation

1. Vascularized pancreatic transplantation Whole or segmental pancreatic transplantation is performed with the venous anastomosis anchored to either the portal or the systemic venous circulation. The exocrine secretions are drained either to the jejunum (Roux-en- Y loop) or to the urinary bladder lumen.
2. Islets of Langerhans implantation (still under study).

Complications

- Technical complications include arterial or venous occlusion; and pancreatic duct leakage.
- Rejection.
- Pancreatitis
- Complications of immunosuppression.

Results

One-year graft survival is 45%.

Heart and Lung Transplantation

Indications

Indications of heart transplantation comprise mainly those patients with ischaemic cardiomyopathy beyond revascularization (45%) or idiopathic cardiomyopathy (55%). Several factors are individualized for each patient e.g. poor quality of life, short life expectancy and poor cardiac function (e.g. ejection fraction <20%).

Combined heart and lung transplantation may be done if both organs are damaged as in cystic fibrosis or patients with primary pulmonary hypertension with severe left ventricular dysfunction.

Single lung transplantation may be done for emphysema or pulmonary fibrosis.

Complications

- Complications of immunosuppression, especially infection (cytomegalovirus).
- Graft rejection.
- Graft atherosclerosis.

Bone marrow transplantation

Indications

1. Severe aplastic anaemia
2. Some leukaemias.
3. To strengthen the depleted bone marrow (blood-making system) of a patient weakened by high, potentially curative doses of radiation or chemotherapies.

Graft-versus-host reaction

The transplanted bone marrow cells are immunocompetent. They can recognize the recipient cells as foreign and can, thus, mount an immune response against the recipient. For this reason bone marrow transplantation is confined to HLA-identical siblings.

Technique

- Suppression of the recipient's immune system by high-dose chemotherapy and whole body irradiation immediately prior to grafting. This is done to inhibit rejection.
- Bone marrow is aspirated from a living donor by multiple punctures of the iliac crest.
- Filtration and heparinization.
- Intravenous infusion of the marrow cells to the recipient.

Intestinal transplantation

The main indications of small intestinal transplantation include the conditions of extensive intestinal resections e.g. mesenteric vascular thrombosis, Crohn's disease, radiation enteritis, and volvulus neonatorum or intestinal atresia in infants.

SKIN AND SUBCUTANEOUS TISSUES

Benign lesions of the skin and subcutaneous tissue

Sebaceous (epidermoid) cyst

A sebaceous cyst is a retention cyst that is caused by blockage of a sebaceous gland duct. It is lined by stratified squamous epithelium and contains a foul smelling, white material composed of keratin, epithelial cells and granular debris.

Clinical feature

- Sebaceous cysts are slowly growing.
- They are rarely seen before adolescence.
- The cysts occur most commonly in the scalp, face, neck or scrotum but they can occur anywhere except the palm or the sole of the foot which are devoid of sebaceous glands.
- The cyst forms a small, well defined, cystic swelling which is usually attached to the skin at one point which is the site of the duct. A punctum may be seen (Fig. 12.1).
- The swelling is mobile over the deep structures.
- Sometimes a sebaceous cyst attains a large size.
- The lesion may be solitary or multiple.

Complications

1. **Infection.** This is, by far, the commonest complication, the others are rare. The cyst becomes painful and tender. There is overlying redness and an abscess may form. If the case is early, it may subside by antibiotics, otherwise if an abscess forms, it should be drained and the wall of the cyst curetted. Infection makes the cyst more difficult to excise as it becomes adherent to the surrounding subcutaneous tissue.
2. **Sebaceous horn.** The contents of the cyst may come out slowly and become inspissated in successive layers over the base.
3. **Ulceration.** An infected cyst may undergo ulceration leading to the appearance of an ulcer with raised edges and overgrowing granulations. This ulcer is called "Cock's peculiar tumour" and it may be mistaken for a carcinoma.
4. **Localized alopecia**

Treatment

Complete excision of the cyst with an ellipse of overlying skin containing the punctum is performed to avoid recurrence (Fig. 12.1). The operation can be done under local anaesthesia if the cyst is small.

CHAPTER CONTENTS

- Benign lesions of skin and subcutaneous tissue
 - Sebaceous cyst
 - Dermoid cysts
 - Callosity
 - Corn
 - Wart
 - Papilloma
 - Keratoacanthoma
 - Vascular anomalies
 - Lipoma
 - Neurofibromatosis
 - Naevi
- Precancerous skin lesions
 - Squamous keratosis
 - Bowen's disease
- Malignant neoplasms of the skin
 - Basal cell carcinoma
 - Squamous cell carcinoma
 - Melanoma
 - Malignant skin tumours of vascular origin

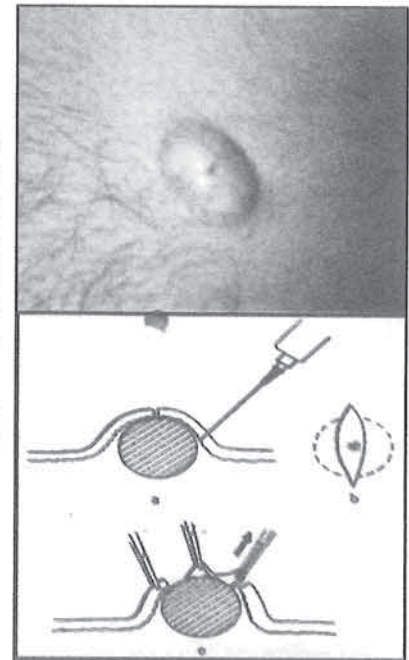


Fig. 12.1. Sebaceous cysts usually affect hairy areas. Notice the punctum. Steps of excision under local anaesthesia.

Dermoid cyst

This is a cyst lined by stratified squamous epithelium and contains sebaceous material. Sometimes hairs or sebaceous glands may grow from the wall of the cyst.

Types

There are several types of dermoid cysts.

1. **Sequestration dermoids.** These are due to subcutaneous inclusion of portions of the surface epithelium along the lines of fusion of cutaneous dermatomes during foetal life. Although present since birth, they may not appear clinically except after few years when the cyst begins to distend. They usually occur at certain sites, e.g. external angular dermoids at the outer angle of the eye (Fig. 12.2), at the root of the nose, around the ear or along the midline of the body. The cyst presents as a well defined, globular, cystic swelling which is not attached to the skin. The underlying bone may be hollowed out and there may be a pedicle connecting the deep aspect of the cyst to the dura mater.
2. **Tubulodermoids** arise from distension of remnants of embryonic ducts such as the thyroglossal duct leading to a thyroglossal cyst or the cervical sinus leading to branchial cyst.
3. **Inclusion dermoids.** These are due to inclusion of the epidermis during closure of a cavity. They include sublingual, suprasternal, intracranial and intraspinal dermoids.
4. **Teratomatous dermoids.** These are benign forms of teratomas. The cyst is lined by squamous epithelium but contains teeth, hairs, bone, cartilage or glands. They occur mostly in the ovary but are occasionally found in the testis or posterior mediastinum.
5. **Implantation dermoids.** These occur secondary to punctured wounds which displace some epithelial cells into the subcutaneous tissues. The displaced cells retain their viability and form a dermoid cyst. These cysts occur mainly in the fingers (Fig. 12.3), palm of the hand or sole of the foot. They are usually small and tense. The overlying skin is sometimes scarred.



Fig. 12.2. External angular dermoid cyst.

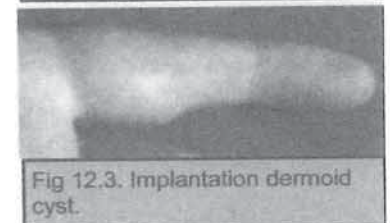


Fig 12.3. Implantation dermoid cyst.

Treatment

The only way of treatment is by surgical excision. In children with a dermoid cyst in the scalp it is better to wait until closure of the skull sutures because some cysts may communicate with the dura. Surgery for a dermoid cyst is more difficult than that for a sebaceous cyst, because a dermoid cyst is deeper.

Callosity

A callosity is an area of superficial thickening of the skin due to continuous friction and pressure. It occurs most often on the toes and sole from wearing ill-fitting shoes and may be also seen on the fingers and hands of manual workers. A callosity is usually not painful and forms a circumscribed yellowish white plaque. Sometimes an adventitious bursa may develop beneath the callosity and if infected, will form an abscess.

Treatment

The patient should avoid the causative agent if possible. The callosity should be shaved with a razor blade and repeatedly painted with an ointment containing salicylic acid.

Corn

A corn is similar to a callosity but a combination of friction and pressure, e.g., tight shoes, produces down growth of a hard horny plug into the corium. The plug causes pressure on the sensory nerve endings leading to much pain.

Treatment is like that of a callosity. Excision and suture are rarely necessary.

Wart

This is due to a virus infection which gains access through an abrasion. It can be transmitted by direct contact. It causes a localized overgrowth of the epidermis and papillae of the skin. Clinically it appears as a small horny projection and may be multiple.

Treatment The options are

1. Curettage and diathermy.
2. Repeated application of glacial acetic acid.
3. Cryosurgery (freezing of the wart) using either liquid nitrogen or nitrous oxide.

Papilloma

This is a benign tumour, usually pigmented which commonly develops in large numbers in the face, arms and upper trunk (Fig. 12.4). There may be a familial trait with autosomal dominant inheritance. The tumour is composed of immature epidermal cells nourished by elongated dermal papillae. Malignant transformation is rare.

Treatment is excision, cryosurgery or cautery.

Molluscum sebaceum (keratoacanthoma)

This lesion is supposed to be due to exposure to the sun. It occurs commonly in the face of middle aged persons. It is composed of keratinizing squamous cells which resolve spontaneously.

Clinically the lesion appears as a firm, rounded reddish papule which enlarges rapidly over a period of 8 weeks producing a rounded, slightly umbilicated mass 1-2 cm in diameter. The centre contains a homy plug covered by a crust concealing a keratin filled crater (Fig. 12.5). Healing occurs by shedding of the homy core. Spontaneous healing takes about six months.

The clinical importance of this lesion is that it may mimic an epithelioma or a rodent ulcer leading to unnecessary surgery. Pathological examination reveals abrupt transition from the normal to the hyperplastic epithelium.



Fig. (12.4) Papilloma



Fig. 12.5. Keratoacanthoma.

Vascular Anomalies

Historically, multiple confusing terms were used to describe vascular anomalies. Terms such as "cavernous" and "strawberry" have been abandoned.

Current nomenclature is biologically based, and includes two major classes

1. **Hemangioma**
2. **Vascular malformations:**
 - Low flow lesions
 - o Capillary malformation
 - o Venous malformation
 - High flow lesions
 - o Arterial
 - o Arteriovenous
 - Lymphatic malformations.



Fig. (12.6A) Haemangioma

Haemangioma

Previously known as strawberry mark. This lesion is a benign proliferative neoplasm of the endothelial lining of vascular spaces. It affects 10% of white infants and the female to male ratio is 3:1. The majority of lesions affect the head and neck area. The lesion usually presents by the following 3 stages

- (a) Stage of proliferation shortly after birth a pale or reddish patch appears and increases in size over the next months (Fig. 12.6A,B).
- (b) Involution phase usually starts at the end of the first year, central pallor appears and the colour of the lesion slowly fades. There is progressive decrease in the size of the lesion. This stage continuous until the age of 10 years.
- (c) Involved phase the skin ultimately is normal in about 50% of patients. Some residual skin changes as discolouration, scarring or telangiectasia may persist.



Fig. (12.6B) Haemangioma of the auricle

In certain circumstances haemangioma may cause problems

- (a) A peri-orbital haemangioma may encroach on the visual field and may lead to amblyopia.
- (b) Haemangioma in the parotid gland may lead to auditory canal obstruction.
- (c) Large cutaneous or visceral haemangiomata may cause congestive cardiac failure.
- (d) Large visceral haemangiomata may lead to entrapment of platelets resulting in thrombocytopenia.

Treatment

1. Reassurance of the parents and observation are recommended as the appearance of the skin after involution may be better than the scar following excision.
2. In those circumstances where it is necessary to interfere the following are helpful
 - a. Oral or intralesional corticosteroids.
 - b. Laser photocoagulation.
 - c. Surgical debulking.

Vascular Malformations

Are due to structural abnormality in the blood vessels due to embryological error. They may be present at birth, but often are noticed later. They grow proportionately with the child and do not regress or involute.

Diagnostic imaging

1. Doppler ultrasound can differentiate high-flow from low-flow lesions.
2. MRI and/or magnetic resonance angiography (MRA) is the most useful study for assessing the extent of the lesion and the degree of involvement of surrounding structures.
3. Angiography is useful in some cases.

- (A) **Capillary malformation (Port wine stain):** This lesion is present since birth and it does not undergo involution. The lesion is dark purple in colour and is not raised above the surface (Fig. 12.7). Pressure causes blanching but the colour returns immediately after release of pressure. Sometimes, the lesion takes the distribution of one of the branches of the trigeminal nerve, but the lesion does not cross the middle line. Sometimes, a port wine stain of the face is associated with similar lesions in the meninges (Sturge-Weber syndrome).



Fig. (12.7) Portwine stain

Treatment

- (a) Pulsed dye laser is the treatment of choice during childhood. It requires general anesthesia and multiple sessions may be needed.
 - (b) YAG laser treatment during adulthood; results are not as favorable as in children.
- (B) **Venous malformation** This lesion is similar to haemangioma in its clinical picture but it usually appears later. As it is a malformation, it does not undergo involution like a haemangioma.



Clinical picture and MRI of venous malformation of the gluteal rea

Treatment

1. Compression therapy may relieve pain and edema.
2. Percutaneous sclerosis may be performed with hypertonic saline, 100% alcohol or sodium tetradecyl sulfate. There is a high recurrence rate; multiple sessions may be required.
3. Surgical excision Preoperative embolization or sclerosis facilitates intraoperative hemostasis.
4. Interstitial laser coagulation.

- (C) **Arterial malformations (cirroid aneurysm)** Arterial malformations (AMs) represent abnormal development of arterial structures, including stenosis or hypoplasia, duplication, and/or tortuosity. They occur most commonly in the scalp (Fig. 12.8) especially in the temporal or occipital regions and may involve the underlying bone. The lesion appears as soft, compressible and pulsating swelling with a marked bruit. Ulceration of the overlying skin may lead to serious haemorrhage as the bleeding is arterial. **Treatment** is difficult as the swelling is supplied by multiple feeding arteries which have to be ligated. Preliminary embolization of the feeding vessels may be tried.

- (D) **Arterial venous malformations** These are high-flow lesions characterized by abnormal connections between arteries and veins without an intervening capillary bed. The lesion is present at birth but may not become evident until late childhood. AVMs may be localized or diffuse. Localized AVM presents by a localized swelling with increased surface temperature. There may be palpable thrill or murmur. Generalized AVMs may cause growth disturbance or skeletal distortion.

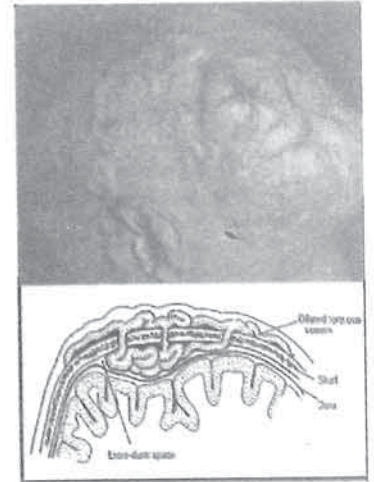


Fig. (12.8) Arterial malformation

Treatment

- Ligation or embolization of feeding vessels often results in rapid enlargement of collateral vessels increasing the size of the lesion.
- Excision Preoperative angiography and embolization of feeding arteries followed by excision next day. Extensive coverage by a free-flap may be needed.

Hereditary haemorrhagic telangiectasia is an autosomal dominant disease characterized by multiple cutaneous, visceral and mucosal AVMs.

- (E) **Lymphatic malformations (LMs)** are classified as microcystic or macrocystic and may have a component of venous malformation. The head and neck region or the axillae are affected in the majority of cases. Neck masses may extend into the mediastinum or prepectoral area. LMs are the most common cause of congenital macroglossia, macrocheilia (lip enlargement), and macrotia. Skeletal involvement may cause distortion.

Treatment

1. Percutaneous aspiration followed by sclerosis may provide short-term improvement; usually requires additional treatment.
2. Surgical excision often requires multiple, staged procedures. It is technically easier as child grows; consider waiting until at least 3 years of age.

Lipoma

This is a benign tumour that is composed of fatty areolar tissue arranged in lobules. A lipoma is enclosed in a thin fibrous capsule, but can be enucleated easily from within this capsule. Sometimes, the tumour contains excess fibrous tissue (fibrolipoma), angiomatous tissue (angiolipoma) or myxomatous tissue (myxolipoma). A lipoma grows very slowly and it is common to find a patient who has the tumour for ten or more years.

Lipomas may present as

1. A solitary well defined swelling.

2. Multiple lipomatosis, the limbs or the trunk are the seat of multiple lipomas (differential diagnosis of multiple swellings).
3. Diffuse lipomatous deposits. These can occur in certain areas, e.g. patients with myxoedema have supraclavicular fatty deposits, elderly persons may develop lipomatous deposits below the chin and sometimes females may develop painful fatty deposits in the thigh (Dercum's disease).

Types

Solitary lipomas are classified according to the site of origin into

1. Subcutaneous lipomata These are the commonest (Fig. 12.9) and they have the following characters
 - a. Slowly growing tumour in the subcutaneous tissues.
 - b. The tumour is painless and is not tender.
 - c. Lobulated surface which may be attached to the skin at multiple points.
 - d. The consistency is soft, although some lipomata especially in warm weather may give pseudofluctuation; this is due to mobility of the tumour in its bed and because fat at warm temperature may undergo liquefaction. Trial of aspiration fails to get fluid out.
 - e. It has a well defined slippery edge due to movement of the tumour inside its capsule.
 - f. The swelling is mobile over deep structures.
2. Subfascial lipomata occur under the deep fascia. This type is difficult to diagnose. It is not attached to the skin and it does not have a slippery edge.
3. Intermuscular lipomata are difficult to diagnose as the swelling is masked by the overlying muscles. These lipomata are fixed deeply and may become more prominent if they are pushed out of the muscle, or disappear if they are drawn into the muscle when it contracts. An intermuscular lipoma has to be differentiated from a fibrosarcoma. The latter is hard in consistency and grows rapidly.
4. Submucous lipomata can arise in the larynx, stomach or intestine. A submucous lipoma in the larynx may cause respiratory obstruction. A submucous lipoma in the intestine may initiate intussusception causing intestinal obstruction.
5. Parosteal lipomata arise in relation to cranial bones and cause erosion of the underlying bones.
6. Extradural lipomata are sometimes found within the spinal canal and may cause paraplegia.
7. Intra-articular lipoma.

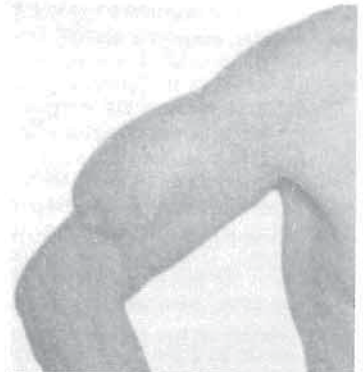


Fig. 12.9. Subcutaneous lipoma.

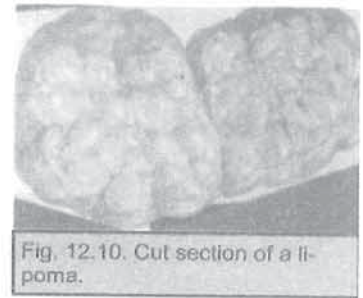


Fig. 12.10. Cut section of a lipoma.

Gross appearance

The tumour has a false capsule around it. It has a lobulated surface and a yellowish colour (Fig. 12.10). Complications are rare and include

1. Degenerative changes leading to liquefaction and calcification,
2. Malignant transformation is very rare, but it can occur in a retroperitoneal lipoma.

Treatment

A lipoma is usually a very innocent tumour which does not cause any problem except in certain situations, e.g. a submucous lipoma in the intestine, or an extradural lipoma in the spinal canal. A subcutaneous lipoma is usually excised for cosmetic reasons. The operation is easy and depends on enucleation of the tumour from inside its capsule.

Neurofibromatosis (Chapter 18)

Benign lesions of melanocytes (naevi)

Melanocytes develop from the neural crest and then migrate to lodge in between the basal cells of the epidermis. Their proportion to the basal cells is constant as 110 to 15 and their number per unit area of the skin is fixed irrespective of race or skin coloration. Differences in skin colour are due to the amount of melanin granules. Melanocytes form melanin pigment and transfer it to the adjoining keratinocytes through their dendritic processes. Melanoma is a true malignant neoplasm that arises from melanocytes. It will be discussed later in this chapter. Various hamartomas can also arise from the melanocytes.

Types (Fig. 12.11)

1. **Lentigo.** Melanocytes replace the basal layer of the epidermis over a certain area. Histologically they appear as rounded cells containing fine granular brown melanin pigment. Clinically it appears as a fault spot varying in colour from pale brown to black.
2. **Junctional pigmented naevus.** More proliferation of melanocytes occurs, leading to small nodules of the epidermis which bulge downwards into the dermis. Clinically a junctional naevus looks like a lentigo.
3. **Compound pigmented naevus.** Eventually the basement membrane is disrupted and some melanocytes pass to the dermis. These melanocytes lose their capacity to form melanin and they are called naevus cells. In the dermis a varying number of macrophages containing melanin pigments are present. These lesions are raised above the surface and vary in colour from pale brown to black. Sometimes dark hairs are seen growing from the surface of the lesion. Rarely, an extensive area of the skin is replaced by such hairy pigmented naevus [giant hairy naevus] (Fig. 12.12).
4. **Intradermal pigmented naevus** Around the age of puberty, in the majority of instances junctional activity ceases, and the naevus cells in the dermis undergo maturation. The lesion is called intradermal naevus and may remain as such for life. Most pigmented naevi of adults are of this type. Clinically they look like compound pigmented naevi (Fig. 12.12).

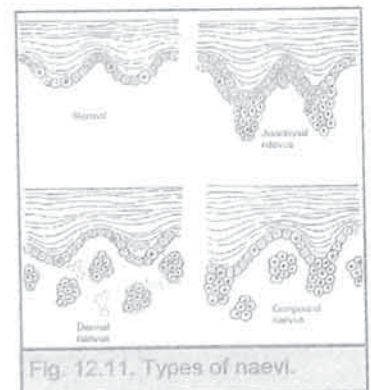


Fig. 12.11. Types of naevi.

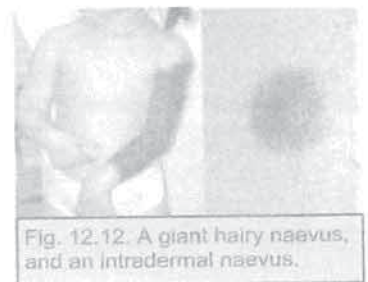


Fig. 12.12. A giant hairy naevus, and an intradermal naevus.

Can pigmented naevi turn malignant?

1. The giant hairy pigmented naevi can turn malignant and give rise to metastases.
2. After puberty any pigmented naevus with junctional activity has the potential to undergo malignant changes, but this is rare.

Indications for surgical excision

1. For cosmetic reasons.

2. If they are subjected to repeated trauma, e.g. during shaving.
3. If there is suspicion of malignant transformation (see under melanoma).

Adequate surgical excision should be performed and the specimen should be examined histologically.

Precancerous skin lesions

Squamous keratosis (actinic or senile keratosis)

These entities are identical and present by dry, rough, inelastic and irregularly pigmented areas of skin. Histologically there are areas of hyperkeratosis, nuclear pleomorphism and increased mitosis with irregular cellular differentiation.

Squamous keratosis is the most important precursor of invasive squamous cell carcinoma.

Bowen's disease

It arises in non exposed skin. It forms sharply demarcated, rounded, reddish patches which enlarge slowly. There is marked epidermal hyperplasia with cellular de-differentiation. This disease is usually treated for a long time as psoriasis.

Malignant neoplasms of the skin

Basal cell carcinoma (rodent ulcer)

This is a locally malignant lesion which arises from the basal cells of the epidermis or from the equivalent cells of hair follicular and sweat or sebaceous glands.

Incidence

The disease is more common in males above the age of 40 years. It is the commonest malignant lesion of the skin.

Aetiology

1. The most important predisposing factor is prolonged exposure to the ultraviolet rays of the sun. That is why the disease is more common in farmers, sailors and country-men living in sunny areas. Persons with light coloured complexion are more liable to the disease than those with dark skin as the presence of melanin pigment protects against the effects of ultraviolet rays.
2. Albinism and xeroderma pigmentosa (Fig. 12.13) predispose to multiple basal cell carcinomata all over the body.
3. Ionizing radiation.
4. Patients receiving immunosuppression are more liable to develop basal cell carcinoma.



Fig. (12.13) Xeroderma pigmentosa

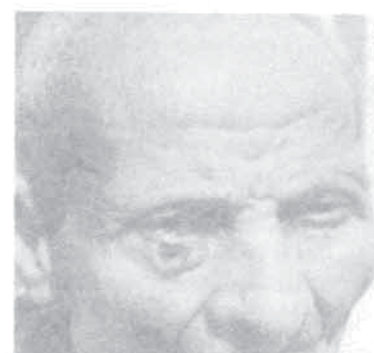


Fig. 12.14. Classic rodent ulcer with rolled in beaded edge.

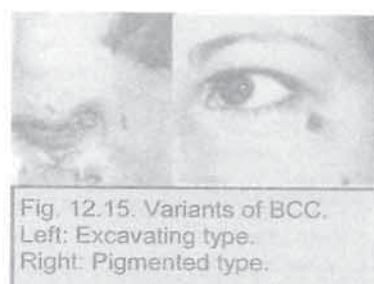


Fig. 12.15. Variants of BCC. Left: Excavating type. Right: Pigmented type.

Pathology

Gross appearance

1. Site. 90% of the lesions occur in the face especially above a line from the lobule of the ear to the angle of the mouth. The commonest sites are the outer or inner canthus of the eye and the nasolabial fold.
2. The lesion usually starts as a small nodule covered by thin epidermis. The nodule ulcerates after sometime with serous discharge and bleeding. Healing sometimes occurs in one area of the lesion while further ulceration occurs. At this stage the patient is usually treated by a dermatologist who notices that this ulcer never really heals.
3. Later the patient usually presents by an ulcerated mass, the edge of the ulcer is rolled-in like the tyre of a car and is beaded. This is the characteristic rodent ulcer that is shown in Fig. 12.14. The rate of growth is very slow and after about one year the lesion may be only one centimeter in diameter (compare with epithelioma). The floor is red and granular and often covered with a dry crust or scab.
4. Another variety of basal cell carcinoma is called the excavating type (Fig. 12.15) where the ulcer erodes deeply into the underlying structures leading to destruction of the nose and infiltration of the nasal sinuses.
5. Other less common types include the cystic, the pigmented [containing melanocytes] (Fig. 12.15) or the flat superficial spreading type which presents as a red scaly patch. The last type resembles psoriasis or eczema.

Microscopic appearance

The tumour cells are arranged as nests or sheets. The outer layer of these groups of cells is composed of low columnar cells arranged side by side like a palisade. Inside this layer the cells are polyhedral with large basophilic nuclei (Fig. 12.16). There are no intercellular bridges. There is no tendency to keratinization and mitotic figures are uncommon. The stroma shows infiltration by lymphocytes. The slow rate of growth of the tumour is due to the long time the cells take to complete one mitotic cycle.

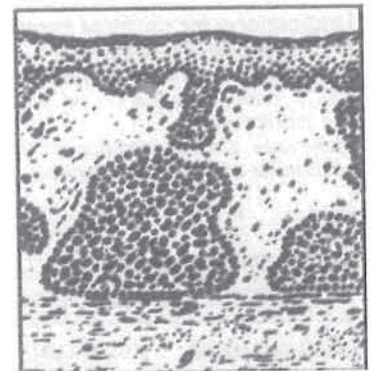


Fig. (12.16) Histology of BBC, characteristic palisading is caused by the side by side arrangement of the outer taller nuclei of cell groups.

Spread

Direct spread to the surrounding and underlying structures is the only mode of spread. Neither lymphatic nor blood spread occur. If the draining lymph nodes are enlarged, this is usually due to secondary infection or the presence of a focus of basosquamous carcinoma.

Treatment

Before treatment the diagnosis should be confirmed by a biopsy. If the lesion is small, excision biopsy is performed, otherwise a small piece of the edge with an area of adjacent skin is excised. Two main lines of treatment are available.

Radiotherapy. Basal cell carcinoma is very radiosensitive, The dose is fractionated over several weeks to diminish the scarring and necrosis. Radiotherapy is contraindicated if there is involvement of an underlying cartilage or bone or if the lesion is near the eyes.

Surgery is indicated for

1. Small lesions as it is easy to close the defect.
2. Infiltration of underlying cartilage or bone as the bone cells will protect the malignant cells from the effect of radiotherapy, Furthermore, radiation is likely to caused troublesome necrosis of the involved bone and cartilage.

3. Radio resistant lesions.
4. Recurrence after previous irradiation.

Surgery should include an adequate safety margin regarding the surface and the depth. A safety margin of 0.5cm is adequate. The defect created after excision can be closed by primary suture or by various plastic rotational flaps.

The pathologist should check that an adequate safety free margin has been excised, and if not, revisional surgery or radiotherapy should be applied.

The prognosis is excellent and provided that the lesion has been excised completely, the cure rate is 100%. Recurrence of the lesion is due to leaving behind foci of malignant tissue and this is more liable to occur if there is infiltration of bone or cartilage. Other possible lines for treatment include cryosurgery and local application of cytotoxic drugs as 5FU ointment (Effudex).

Squamous cell carcinoma (epithelioma)

Incidence

The tumour is more common in males especially elderly persons.

Aetiology

1. Prolonged exposure to ultraviolet rays of the sun (as in rodent ulcer). The tumour occurs more in the upper part of the face, the lower lip, and the dorsum of the hands. Sometimes, the tumour arises at the mucocutaneous junction.
2. Previous irradiation.
3. Albinism and xeroderma pigmentosa.
4. Longstanding irritation of the skin as in chronic granulomas, chronic ulcers, osteomyelitis, sinuses, and old burn scars. A carcinoma arising on the top of a scar is called Marjolin's ulcer, and there is usually a delay in its diagnosis.
5. Prolonged exposure of the skin to carcinogenic agents as polycyclic hydrocarbons, coal tar derivatives, or mineral oils.
6. Immunosuppression.



Fig. 12.17. Typical squamous cell carcinoma with everted edge.

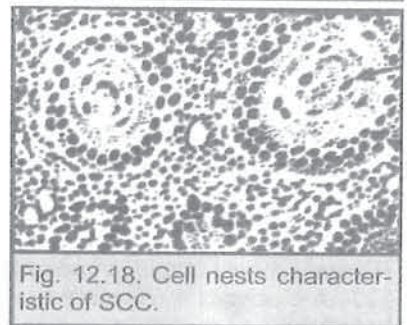


Fig. 12.18. Cell nests characteristic of SCC.

Pathology

Gross appearance

The lesion appears as an ulcerated mass which grows rapidly in contrast to basal cell carcinoma. The edges of the ulcer are raised and everted (Fig. 12.17). The floor is occupied by malignant fungating tissue. The base is indurated and it becomes rapidly fixed to the underlying tissues. Secondary infection may occur and there may be blood stained discharge.

Microscopic appearance

The tumour is composed of groups of carcinoma cells infiltrating the underlying connective tissue. As the groups of carcinoma cells enlarge, the older cells in the centre of the group produce some keratin. Eventually they lose their organelles, become flattened and cornified, and come to lie concentrically in the centre of the cell masses giving a very characteristic appearance termed 'epithelial pearls' or 'cell nests' (Fig.12.18). The presence of prickly cells in the tumour and the formation of keratin are two features that

indicate the differentiated nature of the tumour. In undifferentiated tumours the cells are anaplastic with basophilic cytoplasm which provide no cytological evidence of their origin.

Spread

1. The tumour rapidly infiltrates the adjacent structures.
2. Lymphatic spread to the regional lymph nodes. Sometimes the nodes are enlarged as a result of secondary infection. In patients with Marjolin's ulcer, the fibrosis associated with the scarring makes lymphatic dissemination late.
3. Blood stream spread is very uncommon.

Differential diagnosis

1. Keratoacanthoma.
2. Basal cell carcinoma.
3. Malignant melanoma.

Treatment Two modalities of treatment are available.

Surgery is indicated for

1. Small lesions are treated by excision biopsy with an adequate safety margin.
2. Infiltration of cartilage or bone.
3. Radioresistant lesions.
4. Recurrence after previous radiotherapy.
5. Marjolin's ulcer.
6. Block dissection of metastatic lymph nodes.

The safety margin for epithelioma is at least 2 cm except in the face where it is 1 cm.

Radiotherapy The main indication of radiotherapy is for tumours of the head and neck particularly for poorly differentiated lesions. The resulting scar is rather fragile.

Prognosis 90% five year cure rate.

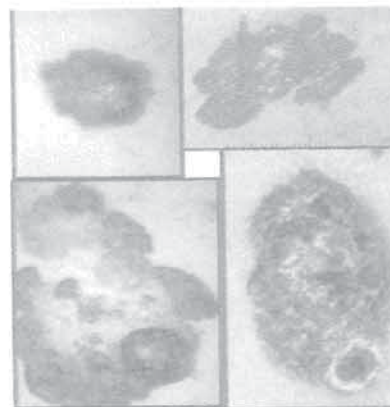


Fig. 12.19. Melanoma. An irregular outline, change of thickness or pigmentation, and ulceration, should raise suspicion of malignancy.



Fig. 12.20. Subungual melanoma.

Melanoma

Incidence

In Western countries the incidence of melanoma is increasing, which may be due to the defective ozone layer. Malignant melanoma is almost unknown before puberty.

Aetiology

1. Prolonged exposure to ultraviolet rays of the sun.
2. Albinism and xeroderma pigmentosa.
3. On top of a benign naevus. Criteria of malignant transformation of a benign naevus (Fig. 12.19) are
 - a. Increase in size or thickness.
 - b. Change of pigmentation.
 - c. Occurrence of itching, tingling, ulceration or bleeding.
 - d. Development of satellite nodules.

Pathology and clinical types

1. Superficial spreading melanoma (64%).
 - They occur in any part of the body, usually in middle age.
 - The lesion is raised above the surface and it has an irregular edge.
 - Malignant melanocytes spread along the epidermis.

2. Nodular melanoma (12-25%).
 - It can occur in any part of the body, usually in the younger age groups.
 - The lesion is raised above the surface, its colour is grey or black, has a smooth surface, and is liable to ulceration.
 - Malignant cells invade the dermis.
 - Prognosis is bad.
3. Lentigo-maligna (Hutchinson's melanotic freckle) (1-15%).
 - The lesion usually occurs in the face in elderly persons.
 - It begins as a flat brown macule which grows very slowly. Some areas may even regress.
 - Malignant cells spread along the basal layer of epidermis.
 - It is the least malignant type.
4. Acral-lentiginous melanoma.
 - This type occurs in the palms, and soles. It may occur beneath the nail (Fig. 12.20).
 - It has a poor prognosis.
5. Amelanotic melanoma. This type has a very poor prognosis.

Malignant melanoma may rarely arise in the eye, in the meninges, or at the mucocutaneous junction as the anal canal.

Prognostic factors

Factors which have a major influence on spread and prognosis are

1. Thickness of the tumour (Breslow classification)
2. Depth of invasion of the skin (Clark's level of invasion) as follows
 - a. Epidermal.
 - b. Dermo-epidermal junction.
 - c. Superficial papillary dermis.
 - d. Deep papillary dermis.
 - e. Subcutaneous tissue.

Spread

1. Direct spread leads to involvement of the subcutaneous tissues and the deep fascia.
2. Lymphatic spread is by permeation or embolism. Lymphatic permeation leads to satellite nodules around the tumour or between the tumour and the regional lymph nodes.
3. Blood stream spread is to the lungs, liver, bones, brain, and skin. Malignant melanoma can spread virtually to any organ. Secondary deposits are usually black.

Differential diagnosis

- Granuloma.
- Pigmented basal cell carcinoma.
- Haemangioma.
- Compound or junctional naevus.

Diagnosis

The only sure method of diagnosis of malignant melanoma is histological examination.

How to take the biopsy?

1. The line of incision should confirm to the possible subsequent excision.
2. Safety margin should be 3 mm.

3. The biopsy should include the whole skin and subcutaneous tissues to allow the pathologist to determine Clark's level and Breslow thickness.
4. Paraffin section is better than frozen section.

Treatment

Surgical excision of the primary lesion with an adequate safety margin of skin and subcutaneous tissue, but not including the deep fascia.

- If the tumour thickness is <1 mm \rightarrow safety margin is 1 cm.
- If the tumour thickness is 1-4 mm \rightarrow safety margin is 2 cm.
- If the tumour thickness is >4 mm \rightarrow safety margin is 3cm.
- If the regional lymph nodes are enlarged and firm, a radical block dissection is performed. If the lymph nodes are not frankly malignant on clinical examination, a fine needle aspiration cytology is performed.
- Prophylactic block dissection is no longer performed.
- Metastases are treated by chemotherapy, interferons and by interleukin-2.

Malignant skin tumours of vascular origin

1. Haemangio-endothelioma.
2. Haemangio-pericytoma.
3. Haemangiosarcoma. The tumour is caused by exposure to vinyl chloride.

Kaposi's sarcoma. The tumour may occur as a sporadic or endemic variety. The latter occurs in certain countries in Africa and it may have a relation to cytomegalic virus (CMV) infection, Kaposi's sarcoma is also one of the manifestations of immunosuppression associated with AIDS.

ARTERIAL DISORDERS

General considerations

Arteries are characterized by the presence of a collateral circulation which under normal conditions is collapsed. This circulation opens whenever the blood flow in the main artery is inadequate for the tissue requirements.

The term ischaemia means diminished blood supply. Ischaemia may be acute or chronic according to the speed of arterial occlusion.

The effects of ischaemia depend upon

1. **The type of artery.** Some arteries have a very efficient collateral circulation to the extent that their ligation may not be followed by serious consequences, e.g., the subclavian artery thanks to the collateral circulation around the scapula. On the other hand, the popliteal artery has poor collateral circulation.
2. **The rate of occlusion of the artery.** Acute ischaemia is much more serious than chronic ischaemia as there is not enough time for the collateral circulation to develop.
3. **The state of the collateral vessels.** Healthy collateral vessels can compensate to some extent the ill effects of ischaemia. On the other hand, if the collateral vessels get thrombosed, the effects of ischaemia will be disastrous.
4. **The general condition of the patient.** The presence of myocardial insufficiency or severe anaemia will exacerbate the effects of ischaemia.

CHAPTER CONTENTS

- General considerations
- Acute limb ischaemia
- Arterial injuries
- Chronic limb ischaemia
- Diabetic foot infection and gangrene
- Aneurysms
- Arterio-venous fistulas
- Extracranial cerebro-vascular disease
- Endovascular surgery
- Thrombolytic therapy

Acute limb ischaemia

Acute ischaemia is a serious condition that occurs due to sudden interference with the blood flow to a limb or an organ. If the treatment is delayed or unsatisfactory, many will lose a limb or even their life.

Aetiology

1. Arterial embolism.
2. Acute arterial thrombosis (either due to complicated atherosclerotic occlusive disease or previous graft thrombosis).
3. Other causes
 - Arterial trauma including intra-arterial drug injection.
 - Aortic dissection (chapter 28).
 - External compression, e.g., a very tight tourniquet.
 - Compartment syndrome.
 - Extensive ilio-femoral DVT (phlegmasia alba dolens).

Arterial embolism

Definition

Embolus is originally a Greek word that means a plug or a stopper. Embolism is the passage of a matter from one part of the circulation to another through a vascular lumen. Arterial embolism is considered the classical example of acute ischaemia.

Causes

The usual source of arterial emboli is a thrombus that is present either in the left side of the heart or in a proximal artery. (Compare with pulmonary emboli whose source is the venous system).

Cardiac sources (common causes)

1. Cardiac arrhythmia (A.F. in 77% of all cases). A.F. is usually associated with mitral valve stenosis or with atherosclerosis that is complicated by a thrombus of left atrium. The latter is the source of emboli.
2. Recent myocardial infarction that causes a mural thrombus.
3. Bacterial endocarditis on top of rheumatic or congenital heart disease, or affecting a prosthetic valve.

Non-cardiac sources (uncommon)

1. A thrombus in an aneurysm. The most frequent are the subclavian, popliteal and abdominal aortic aneurysms.
2. Platelet thrombi on top of an ulcerated atherosclerotic plaque, e.g., in the carotid arteries.

Sites

An embolus in the arterial tree is usually arrested at the sites of bifurcation of arteries where sudden reduction in the size of the arterial lumen occurs. The lower limbs are more commonly affected. The commonest sites of arrest of emboli in descending order of frequency are

1. The bifurcation of the common femoral into superficial and deep femoral arteries (40%).
2. The aortic bifurcation (saddle embolus).
3. The bifurcation of the popliteal artery.
4. The bifurcation of the brachial artery.
5. The bifurcation of the common carotid artery.

Acute arterial thrombosis

Acute arterial thrombosis may occur in the following conditions

1. On top of chronic occlusive arterial disease, e.g., atherosclerosis (commonest) or Buerger's disease.
2. As a complication of arterial aneurysm.
3. As a result of traumatic contusion of vessels.
4. Arterial thrombosis may complicate febrile illness or gastroenteritis especially in children. Dehydration leads to haemoconcentration with consequent thrombosis.
5. Acute graft thrombosis this may occur early due to technical error or late due to progression of the original disease.

Pathological consequences

1. **Secondary arterial thrombosis.** After circulatory arrest widespread distal intravascular thrombosis occurs. Thrombosis may also affect the artery proximal to the site of obstruction.
2. **Compartment syndrome.** Ischaemic muscles get swollen and this, in turn, exaggerates the effects of ischaemia. After revascularization further oedema of the muscles occurs leading to more interruption of the circulation. This muscle compression is more apparent in the muscular compartments of the leg. Early fasciotomy of the muscle compartments can avoid this problem.
3. As a result of stasis of blood in the arterial tree, blood stagnates in the veins draining the limb and deep vein thrombosis may develop.

Clinical consequences

These depend upon the aetiology, site and duration of ischaemia as well as the efficiency of treatment:

1. Complete recovery.
2. Gangrene, which is usually of the wet variety.
3. Chronic ischaemia.
4. Volkmann's ischaemic contracture (chapter 16).

Clinical features of acute ischaemia**Severe acute ischaemia**

Severe acute ischaemia results in the classic symptoms and signs (6Ps)

1. **Pain**
2. **Paralysis or paresis**
3. **Paresthesia**
4. **Pallor**
5. **Pulselessness**
6. **Persisting coldness** "Poikilothermia"

There is poor capillary refilling. Pain is severe and initially the leg is marble white and the veins are empty. After 6-12 hours vasodilatation occurs and the capillaries fill with stagnant deoxygenated blood, resulting in a mottled appearance. Later, the capillaries rupture resulting in fixed blue staining of the skin, "irreversible changes".

Irreversible lower limb ischaemia

1. Fixed colour changes "blue staining".
2. Signs caused by muscle necrosis
 - a. Tense calf.
 - b. Fixed plantar flexion of the foot.
 - c. Bulging anterior leg compartment.
3. Acute paraplegia may occur in case of saddle aortic embolism.

Moderate limb ischaemia It occurs if

1. Embolus does not cause complete occlusion.
2. Pre-existing good collaterals in cases of thrombosis.

The onset is more gradual and the symptoms are less severe.

Investigations

Investigations should be urgent.

Imaging studies

1. **Duplex scan** localizes and identifies the presence of embolism or a thrombus.
2. Arteriography may cause a delay of 2-3 hours. It is, therefore, not done in a threatened limb. Its value is in cases of diagnosed acute thrombosis because it provides information that is essential before doing an arterial reconstruction. This information includes
 - Site of occlusion
 - Proximal inflow, i.e., if there is another proximal arterial narrowing.
 - Flow distal to the occlusion, i.e., distal run-off.
3. Echocardiography detects cardiac sources of embolism. There are two routes, the trans-thoracic and the trans-oesophageal. The latter is four times more informative than the former, because it can detect thrombi in the atrial appendage.

Labarotory studies

- Raised hemoglobin, blood urea nitrogen and creatinine indicate intravascular hypovolaemia due to fluid sequestration in the limb.
- Acidosis and raised creatine phosphokinase and WBCs indicate extensive muscle necrosis.

Differential diagnosis

In addition to differentiation between embolism and thrombosis (table 13.1), other conditions include:

1. Low flow states.
2. Acute occlusion of popliteal and femoral aneurysm.
3. Phlegmasia \square erulean dolens
4. Aortic dissection.

Table (13.1) Differences between embolic and thrombotic acute ischaemia

	Embolism	Acute thrombosis on top of atherosclerosis
History	Arrhythmia or recent myocardial infarction	Claudication
Source of emboli	Usually present	Absent
Radial pulse	Usually irregular (AF)	Usually regular
Skin colour	White	Dusky
Limb nutrition	Normal	Picture of chronic ischaemia
Angiography	Sharp cut-off Minimal collaterals	Tapering stenosis Diffuse atherosclerosis Extensive collaterals

Treatment**General measures**

1. Morphine for pain.
2. IV. fluids to correct dehydration, if present.
3. IV. heparin. Start with a loading dose of 80 IU/Kg followed by a maintenance dose of 18 IU/Kg/hour. The dose is controlled by checking activated partial thromboplastin time (APTT) very 12 hours, which should be maintained at 2.5-3 times the baseline level.
4. Care of cardiac condition
 - a. Oxygen if needed.
 - b. Lab tests, ECG and chest X-ray.
 - c. Digoxin for rapid A.F.
 - d. Lasix for heart failure.

Embolism

Emergency surgery is required to remove the embolus by an operation that is called embolectomy.

- Urgent embolectomy using Fogarty balloon catheter (Fig. 13.1) is the standard method for removal of arterial emboli.
- The operation should be done as long as the limb is viable. The earlier the operation is done, the better are the results because delay leads to obstruction of collaterals by propagated thrombosis.
- The operation can be done under local, general or epidural anaesthesia depending on the patients general condition.

- For embolism of the femoral artery embolectomy is done via a common femoral transverse arteriotomy.
- For aortic bifurcation embolism embolectomy is done via bilateral femoral arteriotomies (Fig. 13.2).
 - The Fogarty catheter is introduced through the femoral arteriotomy and is then passed proximally up past the embolus in the lower aorta.
 - The balloon is inflated and the catheter is pulled out. The balloon sweeps the embolus out of the arteriotomy. The strong arterial blood flow will clear any residual fragments.
 - The Fogarty catheter should also be passed distally to extract any fragments of thrombi and propagated clots.
- For brachial artery embolism embolectomy is done via a brachial artery exposure.
- Arteriography is done on table to ascertain clearance of the arterial tree.
- Heparin should be continued postoperatively until the cardiac condition is assessed and the need for further heparin therapy is determined.
- The source of arterial emboli should be corrected if possible.

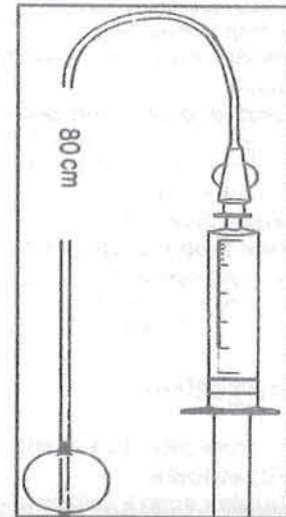


Fig. 13.1. Fogarty catheter.

Acute thrombosis

- Severe ischaemia. Urgent arteriography is performed to plan for emergency revascularization surgery.
- Moderate ischaemia. Urgent arteriography is performed. There is time for the use of thrombolytic therapy. After dissolution of the thrombus elective revascularization surgery is done. Elective surgery carries a better prognosis than emergency surgery in these cases.
- **Thrombolytic agents**
 - Streptokinase is inexpensive but may cause anaphylaxis or be inactivated by antibodies.
 - Urokinase is a direct plasminogen activator but is very expensive.
 - Tissue plasminogen activator (TPA) it is neither pyrogenic nor antigenic and may result in faster lysis. It is also very expensive.

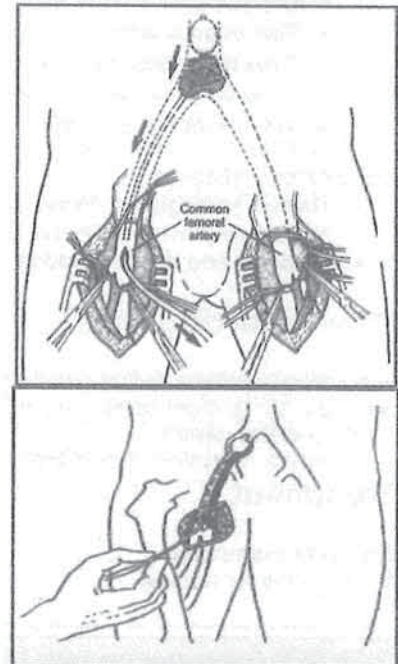


Fig. (13.2) Embolectomy. Removal of an aortic bifurcation embolus

Technique of direct thrombolysis

- At the completion of arteriography the tip of the arterial catheter is embedded in the thrombus.
- The dose of thrombolytic agent differs according to the agent used.
- Monitoring every 6 hours by APTT and fibrinogen titre.

- **Embolic acute ischaemia is usually severe to warrant urgent surgery.**
- **Acute thrombosis may produce less severe ischaemia because previous chronic ischaemia opens collaterals.**

Complications

- Anaphylactic reaction with the use of streptokinase.
- Haemorrhage at the puncture site, GIT or cerebral.

Arterial injuries

Arterial injuries are increasingly common nowadays due to the increased incidence of motor car accidents, civil violence and war injuries. Iatrogenic injuries are also increasing with the introduction of invasive techniques for the diagnosis and management of cardiovascular disorders.

Aetiology

1. Penetrating injuries

- Low-velocity agents, e.g., knives and low velocity bullets. These produce damage to structures in their pathway.
- High-velocity missiles produce widespread damage that may affect a vessel remote from the wound tract.
- Close range shotgun blasts inflict widespread damage through the multiple pellets that penetrate the tissues. These pellets may even embolize in both the arterial and venous systems.

2. Blunt injuries as in road traffic accidents and falls from height. Blunt forces may cause vascular injury through

- Direct injury to major vessels.
- Fractures and dislocations, e.g., suprachondylar fractures of the humerus or the femur or posterior dislocation of the knee.

3. Iatrogenic injuries may follow arterial cannulation for arteriography, cardiac catheterization or dialysis. Injury of the iliac vessels may occur during pelvic operations.

4. Intra-arterial drug injection

Pathological types (Fig. 13.3)

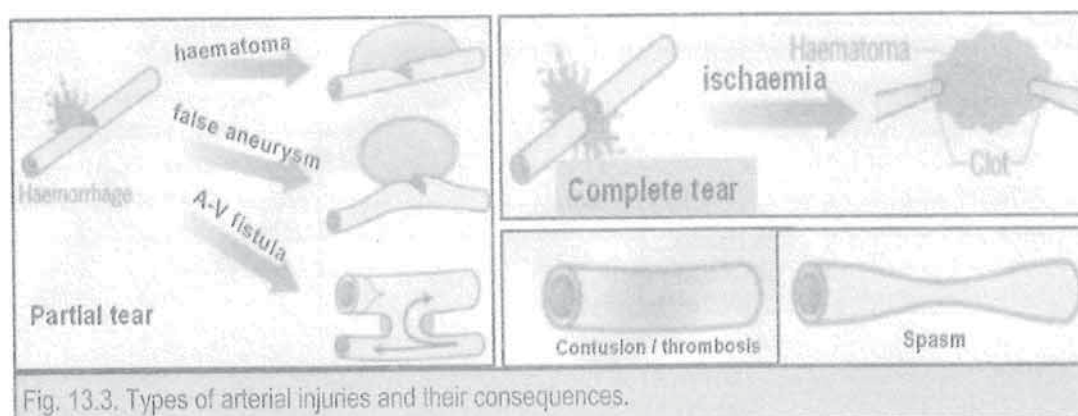


Fig. 13.3. Types of arterial injuries and their consequences.

1. Arterial division. The tear may be complete or partial

- **Complete tear (transection).** The transected ends retract, constrict and thrombose. Therefore, this injury presents with ischaemia rather than haemorrhage.
- **Partial tear.** The elastic coats retract at the site of injury causing widening of the disrupted area. Therefore this injury usually presents with bleeding.

- If such bleeding remains contained within the tissues a pulsating haematoma will develop. This will later form a traumatic false aneurysm (pseudoaneurysm).
 - At other times bleeding may find its way to the exterior through an external wound or to the peritoneal or pleural cavities. In these cases severe life threatening haemorrhage may occur.
 - Less commonly concomitant injury to adjacent arteries and veins may result in an arteriovenous fistula.
2. **Arterial contusion and thrombosis** is the commonest type in cases of closed injuries. Thrombosis occurs over the damaged intima. Patients with contusion present with ischaemia.
 3. **Arterial spasm.** Patients with arterial spasm present with ischaemia. Arterial spasm should not be diagnosed except after exploration of the artery to exclude contusion thrombosis.

Clinical features and assessment

History	Mechanism of injury. Amount and location of blood loss.
General Examination	Vital signs for evaluation of the magnitude of blood loss. General examination to look for other injuries.

Local examination is the basis for diagnosing arterial injury.

- A. **Hard signs.** These are sure signs of arterial injury.
 - a. External arterial bleeding.
 - b. Loss of distal pulses.
 - c. Any of the classic manifestations of acute ischaemia (six Ps).
 - d. Pulsating or expanding haematoma.
 - e. A palpable thrill or an audible bruit heard with a stethoscope at or distal to the area of injury.
- B. **Soft signs.** These are less specific (equivocal) signs.
 - a. Small or moderate sized haematoma that is not pulsating and not expanding.
 - b. Proximity of penetrating wound to a major vascular structure.
 - c. Adjacent nerve injury, producing neurological deficit.
 - d. History of prehospital haemorrhage that has stopped, or presentation with shock that cannot be explained by other injuries. In these cases the patient could have bled from an arterial injury and the bleeding could have stopped before hospital admission.

Investigations

In patients with hard signs

Immediate surgical exploration is indicated without any diagnostic studies. Diagnostic studies in these patients are usually unnecessary and the delay in treatment is dangerous.

In patients with soft signs urgent investigations are needed.

1. Plain X-ray to detect foreign bodies or fractures.
2. Arteriography is the most accurate diagnostic study in these cases. It serves to diagnose the injury and accurately localizes its site. A traumatic arterio-venous fistula is detected by early venous filling.
3. Duplex ultrasonography can be a reliable substitute, provided it is performed by a skilled operator.
4. If the soft sign is "shock" no diagnostic studies should be done. Resuscitation is the first priority. Arteriography can be done in the operating room.

Treatment

Immediate treatment

1. **Control of external bleeding.** Direct local compression of the site of injury is the most effective method at the scene of accident or in the emergency room. The blind placement of clamps into a bleeding wound should be condemned as it can damage adjacent structures and extend the arterial injury. Also, tourniquets should be avoided since they can occlude collateral inflow and lead to greater ischaemic damage. Embedded knives or haematomas should not be disturbed prior to arrival in the operating room as this may precipitate major bleeding.
2. Resuscitation by blood and IV fluids follows the standard measures (chapter 4).
3. **Heparinization.** Systemic heparin is recommended only in patients with isolated vascular injury presenting with ischaemia. Thrombosis can convert an initially reasonable situation into one with risk of tissue loss. However heparin should be avoided in patients with multiple injuries, particularly those involving the CNS, eyes and bones. Heparin in these cases carries an unacceptably high risk, and urgent surgery rather than heparin is the solution.
4. Prophylactic antibiotics should be given early as infection may cause secondary hemorrhage.

Definitive treatment

Time is the most crucial element that determines limb salvage following extremity vascular injury. Therefore all efforts should be directed at restoring normal perfusion as quickly as possible, but certainly within 6 to 8 hours of injury.

1. Arterial repair should be performed only by surgeons experienced in the techniques of vascular repair. If a surgeon with insufficient experience in vascular repair faces a bleeding injured artery during operative exploration of a wound he may ligate the bleeding ends of the artery and transfer the patient urgently to a specialized center. Methods of arterial repair include
 - Transverse tears less than one half of the circumference are directly repaired.
 - Transverse tears more than half the circumference are completed followed by end to end repair.
 - Completely divided artery with a clean cut injury is repaired by direct end to end anastomosis.
 - Longitudinal tears are closed with a patch of vein to avoid lumen narrowing.
 - Arterial contusions. Excision of the whole contused segment and reconstruction with an interposition graft (vein or less often synthetic).
 - Arterial spasm. Local application of papaverine is tried. If not successful forcible dilatation using Fogarty balloon catheter or vessel dilator is done.
2. Local heparin should be used routinely.
3. Injured main veins are repaired before the arteries.
4. Adequate skin covering is essential.
5. Fasciotomy is done in
 - Late cases
 - Muscle oedema
 - Development of swelling or paralysis after revascularization.

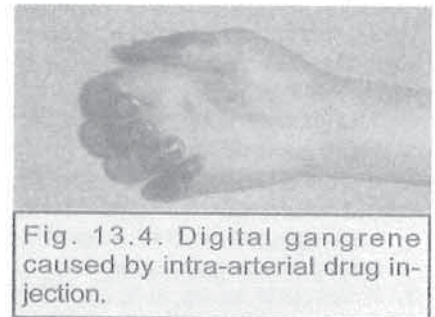
6. In cases of displaced fractures pulses frequently return after reduction (within one hour). If pulses do not return within this period of time, the patient should be explored. One should never assume before exploration that a spasm is present and that it will be relieved with time. This serious error resulted in many limb losses.
7. If vascular injury is found, it should be repaired. Any associated fractures should be fixed by either external or rigid internal fixation prior to the performance of a vascular anastomosis. For simple (closed) fractures internal fixation is permissible. For compound fractures, however, it carries the risk of introducing infection in the bone. In these cases external metal fixation is done.

Intra-arterial drug injection

Drug addicts may wrongly inject drugs into arteries instead of veins. Anesthetists may also wrongly inject thiopentone into an artery during induction of anaesthesia. The most commonly punctured arteries are the brachial and the radial arteries. Thrombosis occurs in the small distal arteries of the digits.

Clinical features

- Burning discomfort extending from the point of injection to the tips of the fingers occurs immediately after injection. This is rapidly followed by severe pain and blanching.
- Oedema develops rapidly and may be severe.
- Coldness and cyanosis soon follow.
- The distal pulses are often normal.
- Digital gangrene (Fig. 13.4) and less commonly total extremity gangrene may then develop.
- Arteriography should not be done as it will not add any more information to the clinical assessment.



Treatment

Treatment should start immediately

- Heparin 10,000 I.U. intravenously, followed by a constant infusion. In cases of intra-arterial thiopentone injection the anaesthetist should not remove the needle from the artery before injecting the heparin bolus intra-arterially. Heparin prevents thrombosis of small vessels.
- Dexamethazone 4mg I.V. every 6 hours. It limits oedema and tissue pressure elevation.
- Low molecular weight dextran (dextran 40) minimizes platelet aggregation.
- Strong analgesics.
- Extremity elevation to reduce oedema.
- Early passive and active exercises.
- Fasciotomy is frequently needed due to chemical myopathy.

Chronic limb ischaemia

This is a slowly progressive arterial obstruction that gives enough time for collaterals to develop and, therefore, gangrene does not occur rapidly.

Aetiology

1. Above the age of 45 years atherosclerosis is the commonest cause.
2. Below the age of 45 years

- In diabetics presenile atherosclerosis is the main cause.
 - In non diabetics
 - In females Raynaud's disease in the upper limbs and arteritis in both the upper and lower limbs.
 - In males Buerger's disease in the lower limbs and arteritis in both the upper and lower limbs.
- Risk factors of atherosclerosis are
- Non-modifiable factors, age, sex, race, family history.
 - Modifiable factors
 - Major Hypertension, diabetes mellitus, hyperlipidemia, smoking.
 - Minor Obesity, physical inactivity, homocystinaemia, CRP.

Atherosclerosis

Pathology

Predisposing factors

- Hypertension, hypercholesterolaemia, and smoking are major risk factors.
- Other risk factors include diabetes mellitus, hypertriglyceridemia, obesity, sedentary or stressful lifestyle, a positive family history and hyperhomocystenaemia.

Pathogenesis of atherosclerosis

1. Subintimal deposition of LDL fatty streaks makes the earliest lesion.
2. Endothelial dysfunction
 - Decreased nitric oxide which counteracts the oxidative stress of free oxygen radicals.
 - Release of pro-coagulant factors.
 - Release of inflammatory mediators.
 - Release of angiotensin II
3. Proliferation of smooth muscle of media due to release of growth factors as platelet derived growth factor and fibroblast growth factor.
4. All the above events lead eventually to the formation of a fibrous plaque leading to arterial narrowing.
5. Complete arterial block may occur due to thrombosis on top of the atheroma or due to haemorrhage in the subintimal plaque.
 - Atheromas most commonly occur adjacent to arterial bifurcations, at the origin of major arterial branches and at sites where an artery passes beneath or through a fascial sling. These factors may cause disturbance of the laminar blood flow and cause abnormal eddy currents.
 - Atherosclerotic lesions have a special distribution. In descending order of frequency they affect the coronaries, cerebrals and carotids, arteries of the lower limbs, the renal, superior mesenteric, coeliac arteries and last are arteries of the upper limbs.
 - Atherosclerosis that causes lower limb ischaemia affects one or more of the following three areas (Fig. 13.5)
 - The aortic bifurcation (aortoiliac disease).

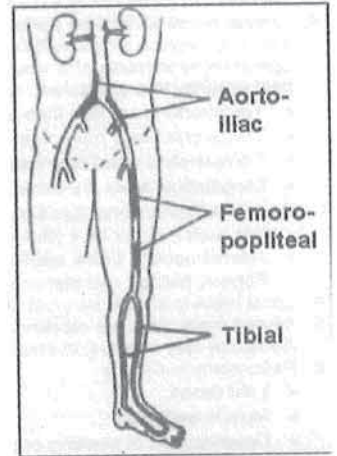


Fig. (13.5): The 3 levels of atherosclerotic arterial disease of the lower limb

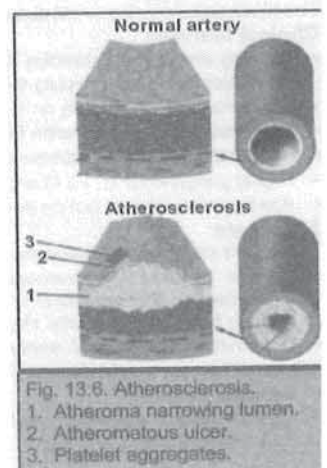


Fig. 13.6. Atherosclerosis.
1. Atheroma narrowing lumen.
2. Atheromatous ulcer.
3. Platelet aggregates.

- The superficial femoral artery (femoropopliteal disease).
- The leg arteries (tibial disease).

Complications (Fig. 13.6)

1. Chronic ischaemia due to gradual arterial occlusion.
2. Acute ischaemia.
 - Ulceration of atheromatous plaques invites platelet aggregation which later detach and cause distal embolism.
 - Acute thrombosis on top of the atheroma leads to complete arterial block.
 - Haemorrhage may occur in the plaque under the intima and is another cause of arterial block.
3. Aneurysm formation.

Clinical features

Symptoms

- **Intermittent claudication.** This is a cramp-like pain that is induced by exercise and is relieved by rest. It affects a certain group of muscles according to the level of obstruction.
 - Patients with aortoiliac disease have pain in the gluteal region, the thigh and the calf, while patients with affection of the superficial femoral artery complain of pain mainly in the calf muscles. Patients with distal arterial disease have pain in the foot.
 - In the early stages of the disease the patient gets the pain after walking for a certain distance (claudication distance) but he can continue his walk. Later on pain gets worse and compels the patient to stop for a few minutes. With progress of the disease, the claudication distance decreases and the time, needed for recovery increases.
 - Ischaemic pain has to be differentiated from venous pain, disc lesions causing sciatica, and pain due to osteoarthritis of the hip or knee joints.
- **Rest pain.** With further progress of the disease, the blood supply of the limb cannot even satisfy the requirements at rest.
 - Ischaemic neuritis causes agonizing burning pain mainly in the foot and toes.
 - Pain is more severe at night when the patient sleeps probably due to further diminution of the blood supply due to elevation of the leg. Another factor is that the warmth of the limb during sleep increases the metabolic demands while the blood supply cannot cope with these increased requirements. Many of these miserable patients spend the night sitting in a chair in an attempt to relieve the pain.
- **Ulceration and gangrene.** In severe ischaemia, superficial painful erosions may occur between the toes, on the dorsum of the foot, or around the malleoli. The end stage of ischaemia is dry gangrene with black mummified skin and tissues.
- **Impotence** may occur in aortic bifurcation block due to diminished blood flow in the internal iliac arteries. Leriche syndrome is a triad of claudication in one or both lower limbs, reduced sexual potency in male patients and diminished or absent femoral pulses.
- Symptoms of atherosclerosis elsewhere may be present, e.g., angina pectoris, cerebral stroke or transient ischaemic attacks and postprandial abdominal pain (intestinal ischaemia).

General examination

- All accessible arteries are palpated to assess the presence of the pulse, any arrhythmias and the condition of the arterial wall. A stethoscope over a stenosed artery may reveal a murmur.

- The blood pressure should be measured.

Local examination

▪ Trophic changes

- Loss of skin appendages as the hairs and sebaceous and sweat glands. The skin becomes dry. The nails become brittle and deformed and lose their luster.
- Loss of subcutaneous fat. The skin becomes thin and the toes become tapered.
- The muscles become wasted and the bones osteoporotic.
- Ischaemic ulcers may develop at the tips of the toes.

- **Temperature changes.** Some degree of coldness will be detected in the ischaemic limb up to a level below but near that of obstruction.

- **Colour changes.** Pallor, cyanosis and sometimes, redness (rubor) may occur. Pallor is due to decreased blood flow into the skin. Cyanosis and redness are due to stagnation of blood in the markedly dilated capillaries under the effect of accumulated vasodilator metabolites. The colour of blood is at first red but it later becomes blue due to extraction of oxygen by the tissues.

- **Absence of pulses.** The pulses distal to the site of occlusion will be absent or markedly weak. A murmur may be heard over the site of the stenotic arterial segment. Normally the dorsalis pedis artery is absent in 10% while the posterior tibial pulse is absent in 2% of individuals.

Simple clinical examination can usually detect the site of arterial occlusion, e.g. in femoropopliteal occlusion the pain affects the calf muscles, trophic, colour and temperature changes are evident in the foot and leg. The femoral pulse is palpable while all distal pulses are not felt.

▪ Special tests

- Test for the capillary circulation. If one presses over the tip of the toe, it becomes pale. Once the pressure is released, the colour returns. In an ischaemic limb the return of colour is slow and is called sluggish capillary circulation.
- Buerger's angle. The patient lies supine and the limb is gradually elevated. The angle at which blanching of the toes occurs is called Buerger's angle. The smaller the angle at which blanching occurs, the more severe the ischaemia is.
- Harvey's venous refilling time. With the patient supine, the limb is elevated to right angle until all veins empty. It is then brought down to the horizontal position. Normally the veins fill in 10-15 seconds, in chronic ischaemia venous refilling is delayed to above 30 seconds.

Clinical staging of chronic limb ischaemia

Stage I	Asymptomatic
Stage II	Intermittent claudication
Stage III	Rest pain
Stage IV	Ulceration or gangrene

Critical limb ischaemia is diagnosed as persistent rest pain, persistent colour changes or the presence of ischaemic ulcers or small gangrenous patches.

Investigations

Laboratory investigations

- Blood sugar.
- Blood picture to detect anaemia or polycythaemia.
- Serum lipids.
- Serum creatinine.

ECG and fundus examination.

Imaging

1. **Doppler flow study.** Ultrasonic waves are directed to the vessel. Their reflection by the moving RBCs can give an idea about the flow pattern and the patency of the artery.
 - Stenosed or occluded segments can be detected.
 - Collateral refilling of the post-stenosed or post-occluded segments can be detected.
 - Measurement of the Ankle/Brachial (A/B) index. By using the ultrasound probe the blood pressure at the ankle and brachial arteries can be measured.
 - Normally A/B index is 1-1.2.
 - Less than 0.9 it denotes ischaemia.
 - Less than 0.7 it indicates severe ischaemia.
 - Less than 0.3 it indicates impending gangrene.
 - Segmental pressures of the different arterial segments can be measured.
2. **Duplex scanning.** This examination can visualize the arteries and detect areas of stenosis or block. It combines the benefits of Doppler plus ultrasound images of the vessels.
3. **Arteriography.** This invasive procedure is only performed if ischaemia is severe enough to raise the need for revascularization procedures. The different techniques of arteriography include
 - a. Direct femoral arteriography. If only one limb is to be studied and provided its femoral pulse is palpable, direct puncture of the artery is done and injection of the contrast material will allow visualization of the whole arterial tree. This technique is losing popularity nowadays in favour of the more informative aortography.
 - b. Aortography. If both lower limbs need to be investigated the contrast material should be injected in the aorta
 - i. Transfemoral aortography (Fig. 13.7). A cannula is introduced into the femoral artery (the pulse is felt on this side), through it a guide wire is introduced. The cannula is removed and then a long catheter is introduced along the guide wire till it reaches the desired level and the contrast material is injected (Seldinger technique). By this technique any artery in the body can be visualized (Fig. 13.8).
 - ii. Transaxillary aortography is only performed if the whole distal aorta is occluded.
 - c. Digital subtraction arteriography (DSA). This technique allows visualization of the arterial tree without arterial cannulation thus avoiding its possible side effects. An X-ray picture is taken and the image is introduced to the computer. An intravenous injection of contrast material is given. The contrast

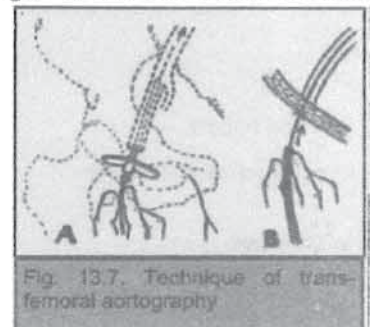


Fig. 13.7. Technique of transfemoral aortography

will travel to the heart and then to the arteries. Further X-rays are taken and the images are computerized. By subtracting the first image from the second an arteriogram can be obtained (Fig. 13.9).

- d. Computed tomographic angiography (CTA) An intravenous contrast and sophisticated equipment afford good quality imaging of arteries. Multiple silices of the arteries can be studied and a post-processing three dimensional (3D) reconstruction allows accurate 3D imaging of arteries or aneurysm. It gives pictures similar to those obtained by conventional angiography. Yet without the need for cannulation of the vessels.

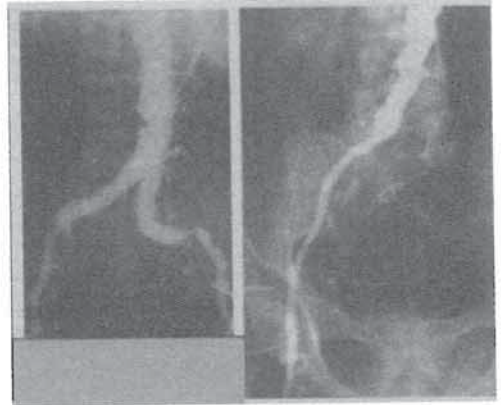


Fig. (13.8) Transfemoral aortography; two example of aortoiliac disease

- e. Magnetic resonance angiography This technique can visualize blood vessels without the injection of any contrast and it can provide a three dimensional image of the examined vessels.

Arteriography provides the following information (Fig. 13.10-11)

- i. Exact site of arterial block.
- ii. State of the proximal arteries.
- iii. Visualization of the artery distal to the block (run off).

These three points are essential if a bypass procedure is planned.

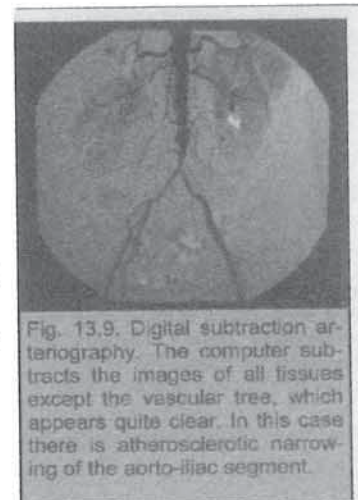


Fig. 13.9. Digital subtraction arteriography. The computer subtracts the images of all tissues except the vascular tree, which appears quite clear. In this case there is atherosclerotic narrowing of the aorto-iliac segment.

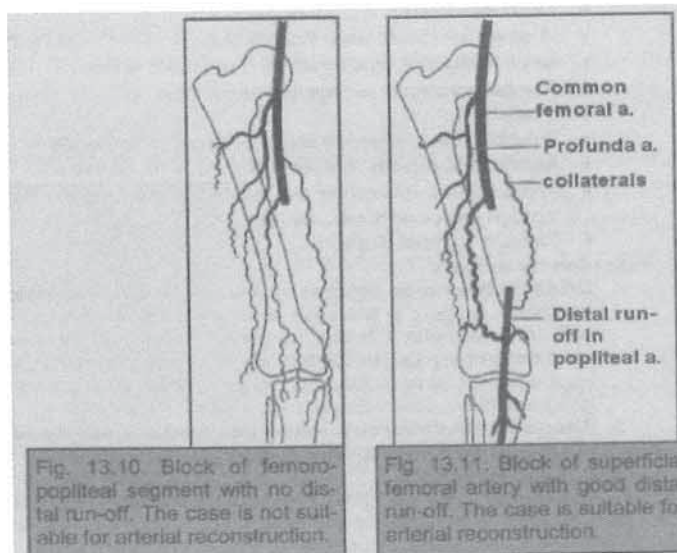


Fig. 13.10. Block of femoro-popliteal segment with no distal run-off. The case is not suitable for arterial reconstruction.

Fig 13.11. Block of superficial femoral artery with good distal run-off. The case is suitable for arterial reconstruction.

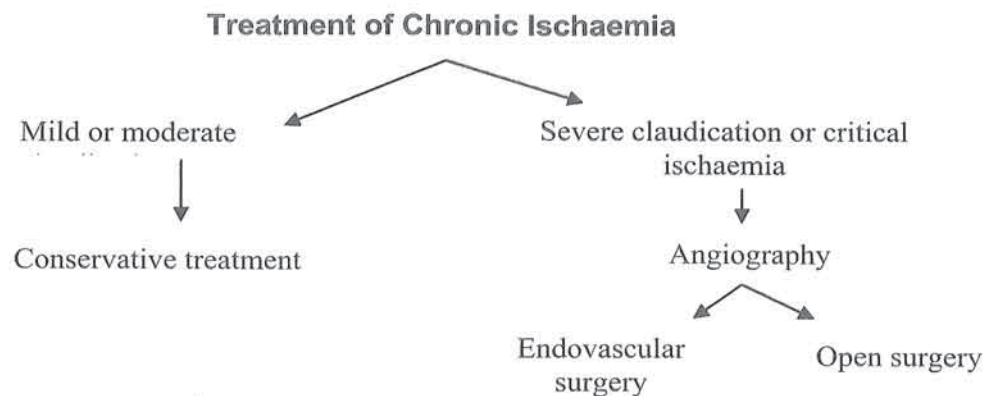


Fig. (13.12): Algorithm of the treatment of chronic ischaemia

Conservative treatment

1. Mild exercises short of causing pain are useful as they help open the collateral circulation.
2. Complete abstinence from smoking.
3. Correction of anaemia as this helps tissue oxygenation.
4. Diabetes mellitus, hypertension and hyperlipidaemia should be corrected.
5. Care of the feet. A patient with chronic ischaemia has a compromised circulation and any minor injury to the foot may precipitate gangrene. The patient is advised to
 - Clean the feet daily and to keep them dry.
 - Avoid feet trauma, e.g., during nail trimming. The patient should not walk bare-footed.
 - In winter he should wear woolen socks to avoid cold injury.
 - Any interdigital fungal infection should be treated.
 - The patient should not apply external heat.
6. Medications
 - Aspirin (150 mg/day) prevents platelet adhesiveness.
 - Statins to correct the hyperlipidemia.
 - Calcium channel blockers.

Indication for surgery

1. Severe incapacitating claudication that interferes with the patient's work and style of life, e.g., a farmer is more likely to need surgery than a clerk.
2. Critical ischaemia as denoted by rest pain and by the presence of ischaemic ulcers or minor gangrene. Surgery in this case aims at saving the limb and is, thus, called limb salvage surgery. A patient with a limited gangrene of a toe can be investigated to check if reconstructive vascular surgery can be performed. A common mistake performed by a beginner is to treat such a patient by amputation of the toe. The surgeon will soon regret this mistake as spread of gangrene may follow the operation.

Endovascular surgery

This is a rapidly growing field of therapy that employs catheter-based technologies to treat vascular diseases. These endoluminal techniques may be applied independently or in combination with conventional surgical procedures.

1. **Percutaneous transluminal angioplasty (PTA).** A special balloon catheter is introduced percutaneously along a guide wire until it lodges in the stenotic segment. The balloon is then inflated and kept for about one minute to dilate the stenosed segment (Fig. 13.37). PTA is indicated for dilatation of short (2 cm or less), significant stenotic segments in otherwise relatively normal arteries. Success rate of about 95% can be anticipated in these cases. Complications include haematoma, A-V. fistula and restenosis.
2. **Intravascular stents.** After balloon angioplasty a stent can be inserted to prevent the elastic recoil of the arterial wall and keep the lumen patent (Fig. 13.38).



Fig. 13.13. Thromboendarterectomy.

Open surgical techniques

There are two types of surgical procedures which can be performed. For both to be successful a good distal run off should be present.

1. **Thromboendarterectomy.** This means removal of the thrombus, intima and the inner media leaving a patent lumen that becomes rapidly endothelialized (Fig. 13.13). Thromboendarterectomy is indicated for a limited number of patients. This procedure is applicable for large arteries with localized obstruction, e.g., a localized narrowing in an iliac artery.
2. **By-pass surgery is more frequently performed.** The idea is to by-pass the obstruction by inserting a graft from the healthy artery above to another healthy artery below the obstruction.
 - For aorto-iliac disease aorto-bifemoral bypass surgery is done using a synthetic graft (Fig. 13.14). Either dacron or polytetrafluoroethylene (PTFE) is used.
 - For superficial femoral artery block the standard operation is femoro-popliteal bypass. The saphenous vein is the best graft for this operation (Fig. 13.15). The graft is anastomosed to a healthy common femoral artery segment above the occlusion and to the popliteal artery below the occlusion. The saphenous vein has valves that allow blood to flow from below upwards only. To overcome this problem one of two techniques are used
 - Reversed saphenous venous graft. A segment of the long saphenous vein is harvested and reversed so that the valves do not interfere with blood flow.
 - In situ saphenous vein graft. The upper and the lower ends of a suitable segment of the long saphenous are used for anastomosis while the vein is left in situ. The valves are destroyed by a valvulotome, and the vein tributaries are ligated.

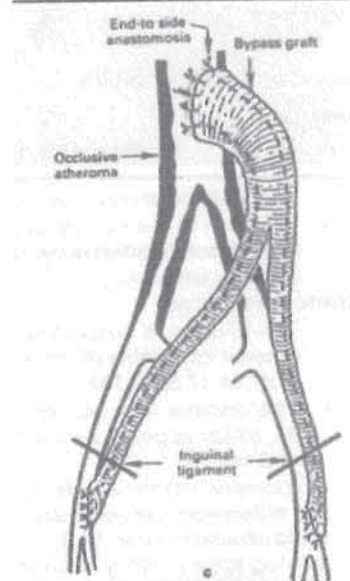


Fig. 13.14. Aorto-bifemoral by-pass using a synthetic graft for aortoiliac atherosclerosis.

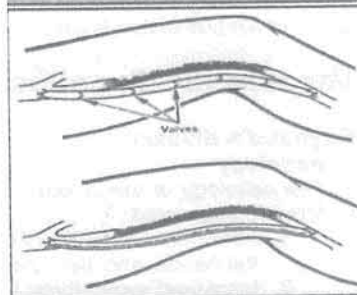


Fig. (13.15) Femoro-popliteal bypass using a saphenous vein graft for femoro-popliteal atherosclerosis

What to do for patients who do not have good distal run-off?

Little can be done for these patients.

- Lumbar sympathectomy does not improve blood supply of the muscles. Therefore, it is mainly indicated when there are cutaneous ulcers or gangrene of the foot. It is also useful for rest pain.
- Intravenous or intra-arterial prostaglandin E may be useful.
- Amputation for gangrene. Dry gangrene is not an emergency. The proper policy is to wait until spontaneous separation occurs or an amputation at the proper level is performed.

Indications for amputation

- Spreading or massive gangrene.
- Spreading infection.
- Severe uncontrollable pain, the patient himself asks for amputation.

Level of amputation

Gangrene complicating atherosclerotic occlusion usually needs major amputation. If the popliteal pulse is palpable, a below knee amputation can be performed, otherwise an above knee amputation may be done according to the surgeon's judgment.

During surgery good vascularity of tissues should be confirmed to guarantee proper healing of the skin flaps.

Thrombo-angiitis obliterans (Buerger's disease)

This is a clinical syndrome of occlusive arterial disease that is characterized by multiple segmental occlusions of small arteries of the lower limbs.

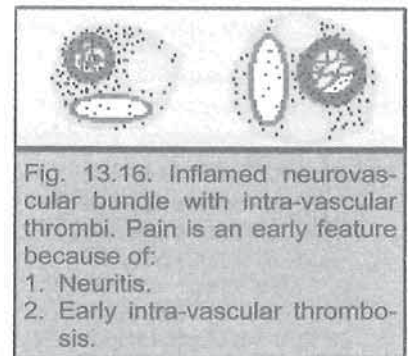
Aetiology

The aetiology is unknown.

- Buerger's disease affects males only
- It starts at an earlier age than atherosclerosis, between 20-40 years.
- The disease occurs exclusively in smokers; it may represent an allergic response to nicotine.
- Interdigital fungus infection is a common association.

Pathology

- The disease has patchy distribution and an episodic course.
- It affects the vessels distal to the popliteal artery.
- The neurovascular bundle shows an inflammatory reaction (panvasculitis and neuritis, (Fig. 13.16).
- The lumen of the artery is obstructed by a thrombus which becomes organized (Fig. 13.16).
- Affection of the nerves by the inflammatory process and the early block of arteries explain the severe pain that is present in many cases.



Clinical features

- The disease is suspected when a young male who is heavy smoker complains of chronic ischaemia in one or more limbs (Fig. 13.17 & 13.18).
- The disease may be preceded by migrating thrombophlebitis, which appears as a small, tender cord along the course of a vein.
- Raynauds phenomenon may be superimposed.

The differences between atherosclerosis and Buerger's disease are illustrated in **Table 13.2**.

Table (13.2) Differences between atherosclerosis and Buerger's disease

	Atherosclerosis	Buerger's disease
Age of start	Elderly	20-40 years
Sex	Commoner in males	Exclusively in males
Aetiology	Main risk factors are <ul style="list-style-type: none"> - Hypertension - High cholesterol - DM 	Excessive smoking
Level of lesion	Aorto-iliac, femoro-popliteal or distal	Distal vessels with patchy distribution
Pathology	Mainly intimal (atheroma)	Inflamed neurovascular bundle & thrombi that block lumen.
Migrating thrombophlebitis	Absent	Usually present
Rest pain	May be present but late	Marked early feature

Arteriography is rarely necessary to confirm the diagnosis. This is because the disease affects distal vessels and distal run off is known to be absent. Therefore there is no possibility for arterial reconstruction, and consequently no need for arteriography.

Treatment

- Smoking must be stopped to avoid disease progress.
- Sympathectomy gives good results.
- Amputation of one or more digits or toes is indicated for persisting pain or gangrene and can be performed adjacent to the line of demarcation with satisfactory primary healing.

Vasospastic disorders

Raynaud's disease

Aetiology

The aetiology of the disease is not exactly known. Certain factors are suggested

1. Abnormal sensitivity of the small arteries and arterioles of the hands, and less commonly the feet, to cold.
2. Increased sympathetic tone.
3. Psychological instability.
4. The presence of cold agglutinins in the blood which cause agglutination of RBCs on exposure to a low temperature.

Diagnosis

Clinical features

For the diagnosis of Raynaud's disease certain criteria should be fulfilled (Fig. 13.19).

1. The disease is bilateral and symmetrical and is much more common in young females, in both hands.
2. The attacks are precipitated by coldness or emotional excitement and are relieved by warmth.

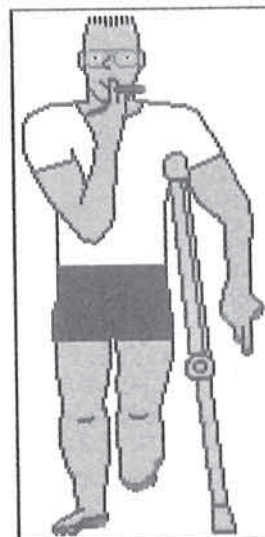


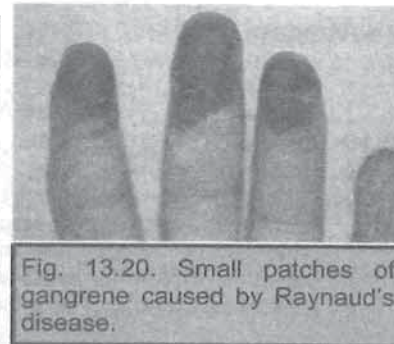
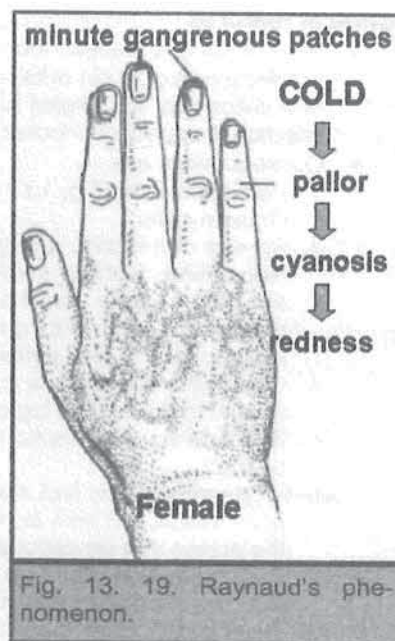
Fig. 13.17. Classic clinical features of Buerger's disease.

- Young male
- Heavy smoker
- Trophic changes and gangrene of toes.



Fig. 13. 18. Trophic changes in the toes in Buerger's disease.

3. The attack consists of 3 consecutive phases
 - a. Pallor; due to spasm of the digital arterioles.
 - b. Cyanosis; due to dilatation of the capillaries which are filled with slowly flowing deoxygenated blood.
 - c. Redness. As the attack passes off, the arterioles dilate and oxygenated blood passes into the dilated capillaries. The attack is accompanied by pain and is called Raynaud's phenomenon.
4. The radial and ulnar pulses are preserved.
5. No major gangrene. Only minute patches of ulceration or gangrene may occur (Fig. 13.20).



Grades of Raynaud's disease

1. First grade presents only with Raynaud's phenomena.
2. Second grade presents with mild trophic changes in the tips of fingers and nails.
3. Third grade presents with gangrene of the tips of fingers.

Treatment

- In the early stages conservative measures are tried.
 - The patient is advised to avoid cold weather and to wear woolen gloves in winter time.
 - Vasodilator drugs.
 - Calcium channel antagonists.
- In severe cases cervico-dorsal sympathectomy is performed. Its immediate results are good but usually the symptoms recur after sometime, but are at least, not severe.

Raynaud's phenomenon

Colour changes similar to those of Raynaud's disease may accompany a large group of organic diseases.

Causes of Raynauds phenomenon include

1. Thoracic outlet syndrome (chapter 24).
2. Certain occupations as typists, pianists and labourers who use vibrating tools.
3. Collagen diseases as rheumatoid arthritis, scleroderma, systemic lupus erythematosus and dermatomyositis.

4. Vascular disorders as atherosclerosis or Buerger's disease.
5. Cryoglobulinaemia.
6. Drugs as chronic administration of ergot-containing drugs for migraine.
7. Atrophic disorders of limbs, e.g. after poliomyelitis.

Treatment should be directed to the original problem. Vasodilators and B-blockers are prescribed.

Diabetic foot infection and gangrene

Diabetic foot infection

This is one of the serious problems which may affect a diabetic patient and which may have disastrous consequences to the limb or even to the patient's life.

Predisposing factors

Diabetic patients are susceptible to serious foot infections due to

1. Peripheral neuropathy. Diminished sensation makes the patients unaware of foot injuries. Motor neuropathy leads to clawing of the toe which leads to undue pressure on the heads of the metatarsals. A callosity forms and underlying infection and ulceration may occur.
2. Vascular affection. One or two factors may impair proper oxygenation and diffusion of nutrients to the tissues.
 - Arterial narrowing by premature atherosclerosis.
 - Microangiopathy.
3. Compromise of the immune system. Both humoral and cellular immunity are disturbed in diabetic patients. The functions of leucocytes (chemotaxis, phagocytosis and intra-cellular killing) are inefficient.

Clinical features

- Most of serious diabetic foot infections start by a trivial trauma or infection, e.g., a pin prick, careless cutting of a nail, scraping of a callosity or interdigital fungal infection. The initial injury or infection is usually overlooked or neglected by the patient.
- Consequences are
 1. Persistent ulcer (Fig. 13.21).
 2. Osteomyelitis.
 3. Spread of infection and gangrene of the foot (Fig. 13.22). Necrotising fasciitis involves fascial spaces and tendon sheaths. Multiple pockets of pus develop and the tissues become necrotic with grey or black sloughs. The patient looks toxic with high pyrexia.
 4. Unless the condition is energetically treated, infection may spread to involve the leg or even the thigh.
 5. Septicaemia and septic shock may develop if pus is not drained.

Diabetic gangrene of the feet may be due to one of the following



Fig. 13.21. Diabetic ulcers of the feet.



Fig. 13.22. Diabetic gangrene of second toe.



Foot infection in a diabetic is serious.

Diabetic foot infection is preventable by following simple foot-care instructions.

1. **Pure infection.** There is no major vascular affection and all the pulses and sensations are intact.
2. **Pure ischaemia.** This is a dry ischaemic gangrene occurring in a diabetic patient with major vascular obstruction.
3. **Neuropathy.** There is loss of pressure sensation and the patient presents with large infected ulcers over pressure areas in the foot, e.g. the heel, or ball of big toe. Later, severe distortion of the bones of the foot occurs with the development of Charcot's joints (chapter 53).
4. **The mixed type.** There is severe infection in addition to major vascular and neuropathic affection.

Prevention

Diabetic patients should be advised to do the following

1. Proper control of diabetes by diet and by medications.
2. Careful trimming of toe nails to avoid their injury.
3. Avoidance of tight foot wear.
4. Early treatment of tenia pedis infection.
5. Daily inspection of the feet to look for wounds and interdigital infections. Peripheral neuropathy allows such lesions to pass unnoticed till infection sets in. Therefore, a breach of the skin should be looked for and treated as early as possible.
6. Avoidance of walking barefooted.
7. Daily feet care by washing, drying, and powdering them.

Foot infection in a diabetic is serious.

Diabetic foot infection is preventable by following simple foot-care instructions.

Investigations

- Blood sugar.
- Plain x-ray may show osteomyelitis, pathological fracture, Charcot's joint or gas in the soft tissue.
- Duplex ultrasound.
- CT angiography.
- MRI to assess the condition of the soft tissues.

Treatment

Diabetic foot infection is a serious problem which should never be treated lightly. Many patients had lost their legs or even their lives due to careless treatment.

1. Hospitalization.
2. Rest in bed and elevation of the foot.
3. Blood sugar is estimated for proper control of diabetes, better by crystalline insulin every 8 hours.
4. Broad spectrum antibiotics are started immediately until the results of culture are available. In many patients anaerobic organisms are involved and should be covered. The dose of the antibiotic and the duration of treatment should be adequate.
5. Drainage of infection and debridement.
 - Under general anaesthesia an experienced surgeon should widely drain all pockets of pus. All sloughs should be excised. A gangrenous toe needs to be amputated leaving the wound open.
 - In some patients infection is so widespread and the patient is in septicaemia. In these cases some form of a major amputation is necessary to save the patient's life. Only when the infection is properly drained, that the patient starts to improve with relief of pyrexia and subsidence of oedema.
 - Repeated dressings, and repeated debridement with drainage of pockets of pus.

- When the wound becomes completely free of infection and contains only viable tissue, a large raw area is considered for plastic skin coverage in order to shorten the recovery time.
- 6. Patients with clinical occlusion of a major artery in the affected limb should have an angiography performed to assess the feasibility of vascular reconstruction. Endovascular management is optimal.

Gangrene

Definition

Gangrene is defined as macroscopic death of tissues that is generally caused by loss of blood supply and is usually associated with bacterial invasion and putrefaction.

Cardinal signs

The five cardinal signs of local death are

1. Loss of pulsation and sluggish capillary circulation.
2. Change of skin colour into blue and later black. The colour does not change by local pressure. This is referred to as "fixed colour changes."
3. Loss of heat.
4. Loss of sensation.
5. Loss of function.

The sentence "Press and See How Colour Fades" is a good reminder of these signs. Patients suffering from threatened gangrene have all the above signs but the tissues are still viable and local pressure causes some modification of colour which returns when the pressure is released.

Aetiology

1. Ischaemic

- Thrombosis, e.g., on top of atherosclerosis (senile gangrene). Gangrene occurs at a younger age in patients who have diabetes, Buerger's disease or other types of arteritis.
- Embolism.
- Vasospastic diseases, e.g. Raynaud's disease and ergotism.

2. Neuropathic Diabetes mellitus, syringomyelia and leprosy.

3. Traumatic

- Direct trauma, due to crushing or pressure (bed-sores).
- Indirect trauma, due to injury of the main vessels.

4. Physicochemical Burns, frost-bite and trench foot.

5. Infective

- Specific infection. Clostridial gas gangrene.
- Nonspecific infections as carbuncle, anaerobic cellulitis, cancrum oris, noma vulvae, phagedena and Meleney's ulcer.

6. Venous gangrene.

Clinical types

There are three types of gangrene depending on the rate of local death and the presence of infection.

1. **Dry gangrene** occurs as a result of slowly progressive vascular disease, as in atherosclerosis (senile gangrene). The gradual slowing of the blood flow permits free evaporation from the affected surface and results in desiccation and mummification of tissues which become hard, dry, wrinkled, shrunken and black (Fig. 13.23).
2. **Aseptic moist gangrene** occurs when the gangrenous process is associated with water logging of the tissues (Fig. 13.24) from (a) sudden occlusion of the main artery

by a ligature or embolus, (b) coincident venous occlusion as in traumatic gangrene. In this form, the part remains of the same size and consistency, but becomes discoloured, being dead- white at first and later purple or greenish-black.

3. **Septic moist gangrene** may be due to infection of sterile gangrene (secondary infective gangrene) or to infection of tissues with virulent organisms which bring about death of the infected tissues (primary infective gangrene). Because of the infection, the part becomes swollen, oedematous, and markedly inflamed. The skin appears moist and blotchy and the epidermis is frequently raised from the surface by bullae filled with serum. The part emits a very offensive odour, and may crepitate due to gas formation from rapid decomposition. There is a marked inflammatory reaction in the living tissues above it, Severe constitutional signs are present and death may supervene rapidly from septicaemia.

Consequences

Gangrene is a serious condition. If untreated, death may occur from progressive spread with severe toxæmia, or blood infection.

Special varieties of gangrene

Senile (atherosclerotic) gangrene

The gangrenous process is usually precipitated by slight trauma, e.g., vigorous paring of a corn or trimming of a nail.

Clinical features

- The onset is often preceded by signs and symptoms of progressive ischaemia, such as intermittent claudication, rest pain and recurrent paronychia.
- Gangrene commences as an area of painful redness in the center of which a slough forms and becomes dry and black.
- The slough may separate leaving an ulcer.
- More often the process spreads gradually.
- The great toe is the commonest site for the onset of the disease which spreads to the foot or neighbouring toes or attacks the other toes independently.
- Pain is marked and causes insomnia and exhaustion.

Traumatic gangrene

1. **Direct traumatic gangrene** is due to local injury, and arises as a result of crush injuries. Crushing leads to moist gangrene and excision without delay is usually indicated. Amputation is performed close to the damaged part to leave a useful limb.
2. **Indirect traumatic gangrene.** Trauma causes damage to the main artery supplying the limb.

Venous gangrene

This is a rare condition caused by extensive thrombosis of the major peripheral veins (phlegmasia cerulea dolens). The high venous pressure causes arrest of the capillary circulation.

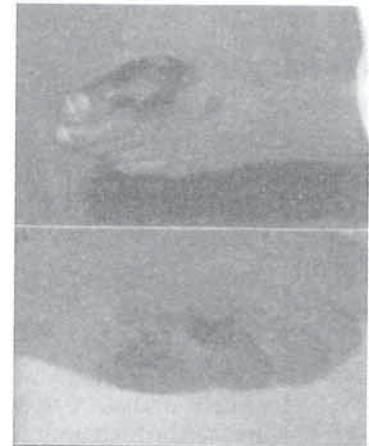


Fig. 13.23. Examples of dry gangrene.



Fig. 13.24. Aseptic moist gangrene.

Treatment

- The limb is elevated and anticoagulant therapy is started.
- Thrombectomy or fibrinolytic treatment should be considered.

Aneurysms

Definition

Pathological definition. An aneurysm is a sac that contains blood and communicates with the lumen of an artery.

Practical definition. An aneurysm is a permanent localized dilatation of an artery, having at least 1.5 times the normal diameter of that given segment.

Classification

Aneurysms can be classified according to

1. Aetiology. Pathological, traumatic and congenital.
2. Structure. True or false.
3. Shape. Fusiform, saccular or dissecting.

Aetiology

Aneurysms are caused by weakness of the arterial wall. Hypertension, if present, helps expanding its size. Atherosclerosis is the commonest cause of aneurysms.

- **Pathological.** Any disease which weakens the arterial wall may lead to aneurysmal dilatation.
 - The commonest cause is atherosclerosis leading to aneurysmal formation at various sites. Common examples are the abdominal aorta and the popliteal artery.
 - Less common causes are cystic medial necrosis, septic emboli of subacute bacterial endocarditis and collagen diseases as Behcet's disease, Marfan syndrome and Ehler's Danlos syndrome. Previously syphilis was a common cause of thoraco-abdominal aneurysms.
- **Traumatic**
 - A blunt trauma to an artery may weaken part of its wall. Later this weak area progressively yields leading to aneurysmal dilatation.
 - A penetrating injury to an artery may cause a small hole in the wall leading to a haematoma surrounding the artery. Later on, this clot is surrounded by a false capsule of organized fibrous tissue and the result will be a false aneurysm (Fig. 13.25).
- **Congenital.** These may occur in the circle of Willis (berry aneurysms, Fig. 13.26) where they may cause subarachnoid haemorrhage. Other sites include the splenic, renal or coeliac vessels.

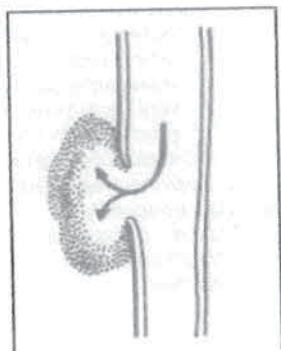


Fig. (13.25) False aneurysm

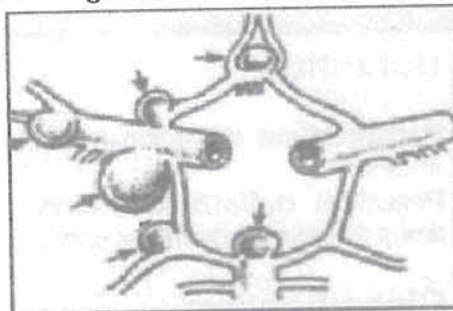


Fig. (13.26) Multiple congenital aneurysms of the circle of Willis

Pathology

Structure of the aneurysm

- True aneurysms. The wall of a true aneurysm is formed of the 3 layers of the dilated artery.
- False aneurysms. A false aneurysm is actually a haematoma communicating with lumen of an artery through a partial tear in its wall. Thus, the wall of the false aneurysm is formed by the fibrous wall of the haematoma (Fig. 13.25).

Complications

- Rupture is the most serious complication. It can produce fatal haemorrhage.
- Distal ischaemia.
 - Thrombosis and occlusion of the aneurysm.
 - Detachment of fragments of this thrombus cause distal embolization. A common example is the occurrence of gangrene in one of the digits due to distal emboli from subclavian aneurysm which develops in patients with thoracic outlet syndrome.
- Infection may lead to rupture and secondary haemorrhage.
- Compression on adjacent structures. As the aneurysm gets bigger, it may compress the adjacent structures. Compression of an adjacent vein may cause obstruction or thrombosis. Compression of a nerve may cause motor or sensory affection. The adjacent bone may also be eroded.

Clinical features

1. Silent aneurysms. Some aneurysms, e.g., of the abdominal aorta are commonly silent. They are accidentally discovered at clinical or ultrasound examination that is done for another reason.
2. Swelling.
3. Symptoms due to compression on adjacent structures.
4. Complications.
5. Local signs of an aneurysm
 - a. A swelling that lies along the line of an artery and can be moved across the line of the artery but not along it.
 - b. The swelling exhibits expansile pulsations (expansile means expansion in all directions, Fig. 13.27). This is the most important sign.
 - c. Proximal pressure on the main artery results in diminution or disappearance of the pulsations.
 - d. Distal compression on the main artery causes the aneurysm to increase in size and to become more tense.
 - e. A systolic thrill may be felt and a bruit may be heard.

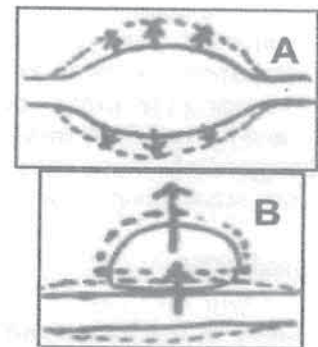


Fig. (13.27) A. Expansile pulsations of aneurysm. B. Transmitted pulsations.

Differential diagnosis

1. A swelling overlying an artery may elicit transmitted arterial pulsations (Fig. 13.27). Pressure on the proximal artery does not change the size of the swelling. If the swelling can be moved away from the artery, the pulsations disappear.
2. A very vascular tumour as an osteosarcoma or metastases.
3. An abscess.
4. A serpentine artery as is sometimes the case with the common carotid.
5. An arterio-venous fistula (see later).

Investigations

1. Duplex scanning is very useful.
2. Angiography may be needed to visualize the distal arteries.
3. CT scan is very accurate in the diagnosis of aneurysms (Fig. 13.28).
4. Magnetic resonance angiography (Fig. 13.29).

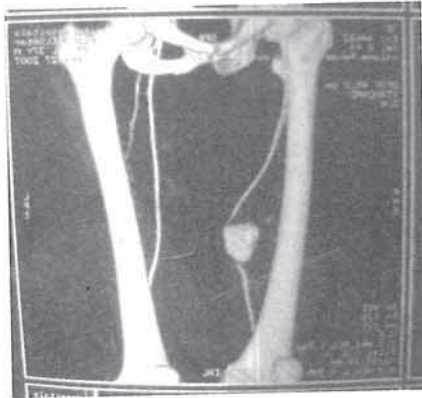


Fig. (13.28) CT angiography showing an aneurysm of the superficial femoral artery



Fig. (13.29) MRI angiography showing an aneurysm of the superficial femoral artery

Treatment

Aneurysms are liable to rupture. So, any aneurysm should be treated surgically.

- The standard line of treatment is excision and graft.
- However, other surgical techniques may be performed
 - Exclusion graft. Insertion of a graft inside the sac of the aneurysm without removal of the sac, this is commonly done in aortic and popliteal aneurysm.
 - Excision with arterial ligation can be done for aneurysms of small arteries as the radial and ulnar arteries.

Abdominal aortic aneurysm (AAA)

This is the most frequent type of aneurysms. It affects the aorta below the origin of the renal arteries in 95% of cases. It may extend to affect the iliacs. Rarely does it extend upwards to involve variable distance of the suprarenal abdominal aorta or the thoracic aorta in which case it is called a thoraco-abdominal aortic aneurysm.

Aetiology

Atherosclerosis is the commonest cause and is responsible for 95% of AAAs.

Clinical features

1. **Asymptomatic aneurysms.** In 75% of patients the AAA is discovered accidentally during a routine abdominal examination (as a pulsatile epigastric mass) or during a radiographic study performed for some other reason (plain X-ray, ultrasonography or CT scan).
2. **Pain is the commonest symptom.** An A.A.A. gradually enlarges and impinges on surrounding structures causing vague abdominal pain. Back and flank pain result from vertebral compression. Large aneurysms can erode the spine and cause severe back pain in the absence of rupture. These patients may be wrongly diagnosed as having lumbar disc lesions.

3. **Symptoms of rupture.** The classic triad of AAA rupture is sudden severe pain, a pulsatile abdominal mass and shock. However, sometimes one or more of the components of the triad are absent in a patient with rupture.
- Acute upper abdominal pain. The abrupt onset of severe pain in the back, flank or abdomen is characteristic of aneurysm rupture or acute expansion.
 - Pulsatile abdominal mass, which is usually tender. It may be masked by obesity or abdominal distension.
 - Shock is present in most cases at the time of presentation. In other cases rupture is so effectively contained within the retroperitoneum that hypotension may be absent at the time of initial examination. If the patient is left untreated, shock may develop at any time.

Investigations

1. **Ultrasonography.** If an AAA is clinically suspected, ultrasonography is the screening test of choice to document or to rule out the presence of an aneurysm. It is rapid, inexpensive, non-invasive and accurate.



Fig. (13.30) 3D CT angiography showing aneurysm of the aortoiliac arteries

2. **C.T scan** (Fig. 13.30,13.31). If repair of an AAA is decided CT scan provides data that are important for surgery especially if endovascular repair is considered. In this respect spiral CT is superior to the conventional axial CT as the former can display the information in multiple planes and allow three-dimensional reconstruction of the aneurysm sac.
3. Magnetic resonance arteriography is a good alternative to CT scan but is more costly.
4. Arteriography. An arteriogram may appear normal despite the presence of a large AAA if the aneurysm is occupied by a thrombus, so angiography is neither used to diagnose AAA nor to assess its dimensions. Angiography is of value if the patient has been suspected to have associated occlusive disease in the iliac, renal, or mesenteric arteries to plan the simultaneous repair of the occlusive disease.

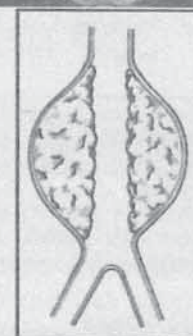


Fig. 13.31. CT scan showing AAA with a thrombus inside.

Natural history of AAA

Rupture is the most frequent and most serious complication of AAA. The risk of rupture is mainly related to the size of the aneurysm. The 5-year rupture rates for untreated AAA of diameters 5cm or more is high, even if asymptomatic.

Treatment

▪ Plan of management

- Immediate surgery for patients with the diagnosis of rupture. These should be taken to the operating room immediately for aneurysm repair. No time should be lost in a surgical ward or in an ICU.
- Urgent surgery for patients with symptoms of acute expansion (severe pain of acute onset but with no leak on C.T. scan).
- Elective surgery for
 - Symptomatic aneurysms regardless of the size.
 - Asymptomatic aneurysms 5cm or more in diameter.

- Regular follow up is indicated for patients with asymptomatic aneurysms less than 5 cm in diameter. Ultrasound is done every 6 months. Aneurysm repair is required if the serial studies show an enlarging aneurysm.

▪ Type of surgery for aneurysm repair

- The standard is conventional open surgery by opening the aneurysm and excluding it by implanting a synthetic graft (Fig. 13.32).
- Endovascular repair of AAA. This means insertion of an endoluminal stented graft through bilateral femoral arteriotomies. This technique is expensive and recurrence of the aneurysm may occur. It is, therefore, indicated for
 - High risk patients who can not tolerate anaesthesia and open surgery.
 - High-risk local abdominal factors, e.g., previous major surgery, intra-abdominal infections, abdominal wall

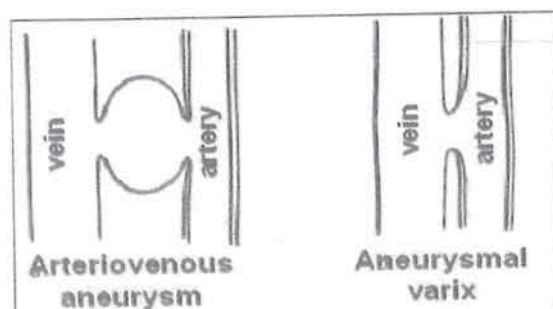
Arteriovenous fistula (A-V fistula)

This is an abnormal communication between an artery and a vein. It may be congenital or acquired.

Congenital arteriovenous fistula (Chapter 12)

Acquired arterio-venous fistula

This is usually secondary to a penetrating injury by a knife or a bullet which induces a communication between an artery and a neighbouring vein. A common site is the femoral triangle, as during angiographic procedures the cannula may injure the vessels leading to A-V fistula. In patients with chronic renal failure who need haemodialysis, an A-V fistula is intentionally made by the surgeon, usually between the cephalic vein and the radial artery. This arterializes the superficial veins of the limb, i.e. they become distended with fast-flowing arterial blood which makes them convenient for puncture and drawing out blood for haemodialysis.



Pathological types of acquired A-V fistula

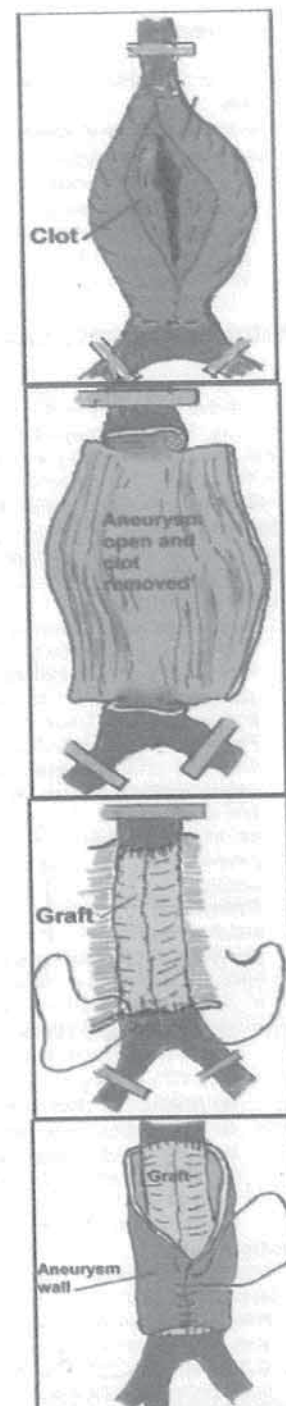


Fig. 13. 32. Repair of AAA.

Pathological types

- Arterio-venous aneurysm. The communication between the artery and the vein is not direct and there is an intervening haematoma which later organizes.
- Aneurysmal varix. The communication between the artery and the vein is direct and there is no swelling between them.

Clinical features

- Local signs. There is a small swelling along the course of the vessels. The striking feature is that the swelling has a continuous thrill over it and auscultation reveals a continuous machinery murmur over the swelling and propagated along the vein.
- Distal signs. The veins become dilated, tortuous, thick walled and pulsating. Oedema, congestion may appear.
- Systemic signs. If the fistula is of a moderate or large size, there will be marked reduction in the peripheral resistance and an increase in the venous return. These will lead to tachycardia, increased cardiac output with high systolic and low diastolic blood pressure (water hammer pulse). Local pressure at the site of the fistula causes marked slowing of the pulse (Branham's sign). A high output large fistula can lead to cardiac failure.

Investigations

- Doppler study can localize the site of the fistula.
- Arteriography will show both the artery and vein at the same time due to rapid venous filling. It may also help localize the site of the fistula.

Treatment

The ideal treatment is by excision of the sac and restoration of continuity of both artery and vein.

The commonest type of A-V fistula is that intentionally induced by surgeons to allow haemodialysis for chronic renal failure patients.

Extracranial cerebrovascular disease

Cerebrovascular disease is the third leading cause of death and is a major source of disability among elderly people. Thrombo-embolic disease accounts for about 75% of strokes; of these, carotid artery occlusive disease is the most important cause.

Pathogenesis

- Embolization is the commonest cause of transient ischaemic attacks (TIA) and ischaemic stroke.
- The embolus may arise from the heart, but more commonly it arises from an ulcerating atherosclerotic plaque at the carotid bifurcation. Ulceration and irregularity of the plaque stimulate platelet aggregation (Fig. 13.33).



Fig. 13.33. Usual source of cerebral emboli.

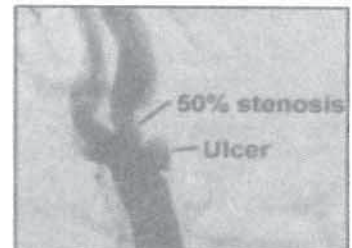


Fig. 13.34. Carotid arteriography showing atherosclerotic stenosis.



Fig. 13.35. Carotid endarterectomy.

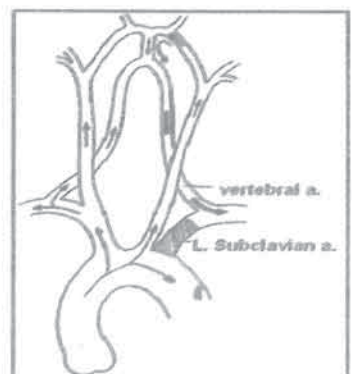


Fig. 13.36. Subclavian steal syndrome.

- If the platelet aggregates embolize to an important vessel in the brain, symptoms occur.
 - If the platelet aggregates break up quickly the symptoms are transient and the patient experiences a TIA.
 - If the embolic fragment persists, it can lead to focal infarction and the patient experiences a stroke.

Clinical features

Patients can be classified into three categories

1. **Asymptomatic patients.** The atherosclerotic lesion in the carotid artery is discovered accidentally by angiography performed for an unrelated condition or by duplex scan carried out because of the presence of a bruit over the carotid arteries or carried out as a screening test prior to major surgical procedures, e.g., coronary artery bypasses.
2. **Patients with transient ischaemic attacks (TIAs).** Transient ischaemic attacks are defined as temporary focal neurologic or visual deficits lasting less than 24 hours and end with complete recovery. TIAs are of sudden onset and resolution is usually within minutes.
 - **Carotid artery TIAs** are caused by deficits in areas supplied by the anterior and middle cerebral arteries. Manifestations include one or more of the following
 - Motor dysfunction as weakness or paralysis of one or both limbs contralateral to the affected hemisphere.
 - Sensory alterations as numbness, loss of sensation or parasthesia in one or both contralateral limbs or in the opposite side of the face.
 - Receptive or motor aphasia if the dominant hemisphere is affected (the left side in 95% of patients).
 - Amaurosis fugax is ipsilateral transient loss of vision described by the patients as a black curtain coming across the eye.
 - **Vertebrobasilar TIAs.** Symptoms include vertigo, ataxia, dizziness, bilateral parasthesias and visual hallucinations.
3. **Patients with stroke.** The patient has residual neurologic deficits that could be minimal or profound depending on the extent of brain damage. A stroke may be fatal.

Investigations

1. **Duplex scan** is the first-line investigation to study the extracranial cerebrovascular arteries in a symptomatic patient. If done by a skilled operator it can give accurate information on the presence and the degree of stenosis.
2. **Arteriography** (Fig. 13.34). Conventional arteriography is associated with a 1—2% stroke rate. It is not suitable as first-line investigation.
3. **CT scan of the brain** is done before operation to define the presence of pre-existing cerebral damage and to exclude other brain pathology.

Treatment Surgery

The operation is carotid endarterectomy. The atherosclerotic plaque at the carotid bifurcation is removed surgically (Fig. 13.35). By removing the plaque the risk of a future stroke is markedly diminished.

Indications of carotid endarterectomy Internal carotid artery stenosis of 70% or greater in a patient with:

1. Carotid artery TIAs.

2. Stroke leaving minimal neurologic deficit.
3. Asymptomatic lesion.

Medical treatment

Indications

1. Less than 50% stenosis even if symptomatic.
2. Patients during the acute phase of a stroke. Revascularization is contraindicated as it leads to haemorrhage in the infarcted brain area.
3. Patients suffering from stroke with poor recovery.

Medications

1. Antiplatelet drugs such as aspirin or ticlopidine. They inhibit platelet aggregation.
2. Stopping smoking and control of diabetes, hypertension and hyperlipidaemia.
3. Full anticoagulation in patients with cardiac embolic disease.

Either medical or surgical treatment is acceptable options in symptomatic patients with 50-69 % stenosis.

Subclavian steal syndrome

In this syndrome cerebral ischaemia is caused by the reversal of flow in the ipsilateral vertebral artery distal to a proximal lesion in the first part of the subclavian artery (Fig. 13.36).

Clinical features

Vertebrobasilar insufficiency occurs on exercise of the upper limb. This is due to increased demand for blood, which the extremity steals from the cerebral circulation through the ipsilateral vertebral artery.

Investigations

1. Duplex scanning
2. Arteriography

Treatment

Surgery. Options include

1. Carotid-subclavian artery bypass
2. Percutaneous transluminal balloon angioplasty (PTA) of subclavian stenosis

Endovascular surgery

Definition

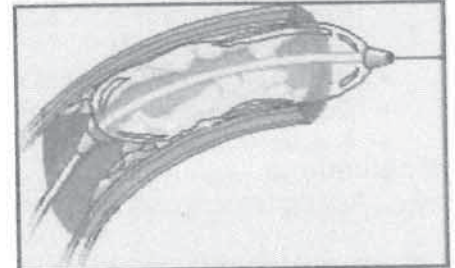
Endovascular surgery is the management of vascular diseases percutaneously through a puncture to deal with a lesion in a remote site.

Classification

- System
 - Arterial
 - Venous
- Purpose
 - Diagnostic
 - Therapeutic

Therapeutic procedures

1. Angioplasty. (dilatation of a vessel).
2. Intravascular stents (insertion of stent to maintain patency of vessel lumen).
3. Thrombolytic therapy.
4. Atherectomy. (removal of atheroma from the wall of an artery).
5. Peripheral laser angioplasty.
6. Endoluminal grafts. (placement of stented grafts inside the lumen of an artery)
 - a. Occlusive disease
 - b. Aneurysms
7. Inferior vena cava filters.
8. Catheter vascular access Implantation of a catheter to access a central vessel for either
 - a. Haemodialysis in chronic renal failure
 - b. Chemotherapy



Balloon angioplasty (percutaneous transluminal angioplasty = PTA, Fig. 13.37)

Indications

These are the same as those for conventional vascular operations.

1. Critical ischaemia as denoted by rest pain and the presence of ischaemic ulcers or minor gangrene.
2. Severe incapacitating claudication that interferes patient's work and style of life.

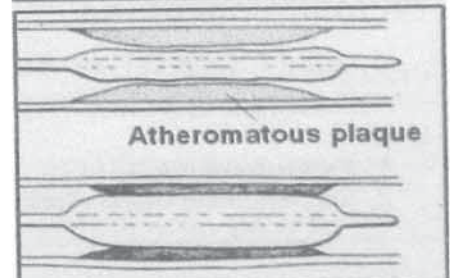


Fig. 13.37. PTA

Ideal lesion

1. At least 5mm beyond origin of an artery.
2. Stenosis < 5cm length.
3. Best in iliac artery.

The technique is also very useful as an adjunctive procedure with surgical revascularization.

Intravascular stent

Aim A stent may be inserted in the same session after PTA to prevent its drawbacks (dissection and elastic recoil) in order to maintain patency of the artery.

Principle

- A metal vascular stent is guided over a guide-wire, endoluminally, and is directed to the arterial segment that has been dilated.
- The stent is carried over an expanding device that is kept closed until the stent reaches the desired site. The device is then opened to expand the stent so that it will fit in the artery (Fig. 13.38).

Types

1. Balloon- expandable stents.
2. Spring-Loaded, self- expanding stents.
3. Thermal memory stents.

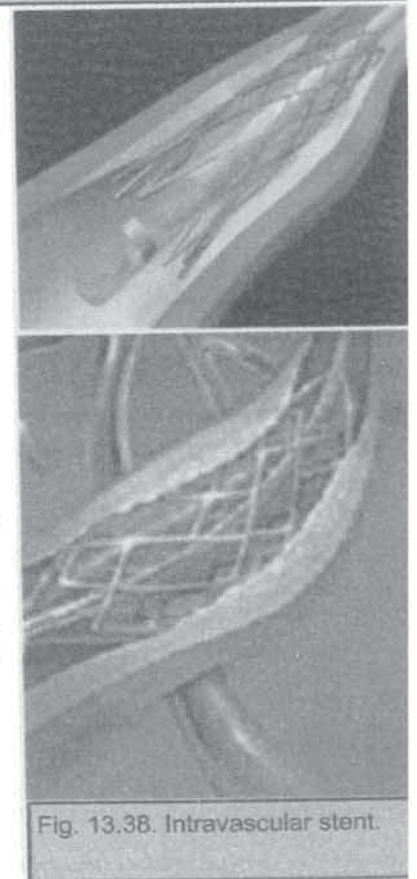


Fig. 13.38. Intravascular stent.

Inferior vena cava filter**Indications**

- DVT or documented pulmonary embolism in a patient who has a contraindication to anticoagulation, e.g., previous intracerebral hemorrhage or history of bleeding peptic ulcer.
- Recurrent pulmonary embolism despite adequate anticoagulation.
- Complications of anticoagulation that forced therapy to be discontinued.
- Failure of another form of caval interruption, demonstrated by recurrent pulmonary embolism.
- Chronic pulmonary embolism in patients with pulmonary hypertension and cor-pulmonale

Endoluminal grafts**Principle**

A combination of a stent with prosthetic graft positioned with a delivery system.

Uses

1. Exclusion of A.A.A.
2. Support of the intima of an artery after balloon dilatation in some cases.

Thrombolytic Therapy

The aim is to inject certain substances which activate the fibrinolytic system leading to dissolution of fresh thrombi in the arterial or venous system. Ideally the fibrinolytic agent should be given as a regional therapy directed into the thrombus via an endovascular catheter.

Indications

1. Acute deep vein thrombosis.
2. Acute thrombotic ischaemia in this situation after dissolution of the thrombus if there is a stenotic segment, it can be treated by balloon angioplasty and/or stenting.
3. Acute coronary occlusion.
4. Occlusion of vascular grafts.
5. Restoring the patency of recently occluded central venous catheters.
6. Pulmonary embolism.
7. Acute embolic ischaemia.

It is better to be done in an ICU under close observation.

The main contraindications include active bleeding, recent stroke, major operations within two weeks, infected bypass grafts and/or infective endocarditis.

The main agents used are **streptokinase**, **urokinase** and **recombinant tissue plasminogen activators**.

VENOUS DISORDERS

Surgical anatomy and physiology of the lower limb veins

There are four anatomically and functionally distinct sets of veins that drain the lower extremities (Fig. 14.1).

1. The superficial veins

These are subcutaneous veins that lie superficial to the deep fascia. They consist of the long saphenous vein on the anteromedial aspect of the thigh and leg, and the short saphenous vein on the posterolateral aspect of the leg, and their tributaries.

The long saphenous vein

Beginning. This vein begins as an upward continuation of the medial marginal vein of the foot.

Course and relations. The vein runs upwards anterior to the medial malleolus then crosses the lower quarter of the tibia obliquely and runs up behind the medial border of the tibia towards the knee where it lies a hand breadth behind the medial border of the patella (an important surface landmark), it then spirals forwards round the medial convexity of the thigh.

The saphenous nerve accompanies the long saphenous vein in the lower part of its course below the knee very closely, and then up to the groin slightly away

from it, till it pierces the deep fascia at the subsartorial canal.

Termination. It ends by passing through the cribriform fascia covering the saphenous opening which lies 3.5 cm below and lateral to the pubic tubercle, where it joins the anteromedial side of the femoral vein.

Tributaries (Fig. 14.2)

1. Four tributaries in the region of the saphenous opening corresponding to the branches of the femoral artery (the superficial circumflex iliac, the superficial epigastric and the superficial and deep external pudendal).
2. The posteromedial and anterolateral veins of the thigh drain a wide area of the thigh and join the long saphenous vein below the saphenous opening.
3. The suprapatellar vein draining the anterior surface of the knee.
4. The anterior and posterior arch veins of the leg which drain a wide area of the leg.

CHAPTER CONTENTS

- Surgical anatomy and physiology of the lower limb veins
- Venous thrombosis- General principles
- Superficial vein thrombosis
- Deep vein thrombosis
- Pulmonary embolism
- Varicose veins of the lower limbs
- Venous ulcers

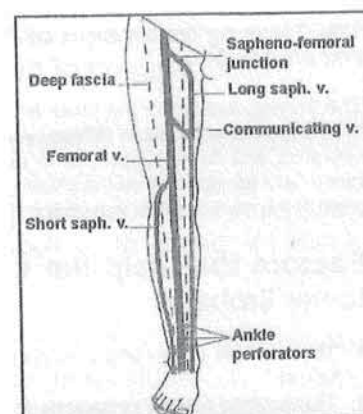


Fig. 14.1. Arrangement of lower limb veins.

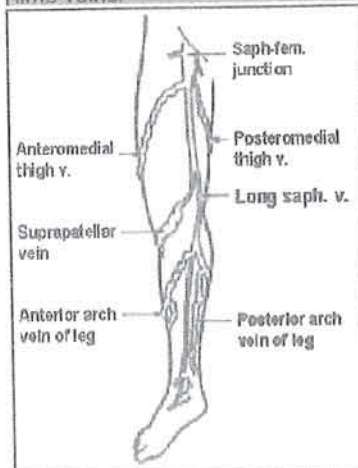


Fig. (14.2) Course and tributaries of long saphenous vein. In this diagram the tributaries are varicose.

The **short saphenous vein** drains the lateral side of the dorsal venous arch and the lateral margin of the foot. It lies with the sural nerve behind the lateral malleolus, then passes upwards in the subcutaneous fat along the midline of the calf and pierces the deep fascia anywhere from the mid calf to the roof of the popliteal fossa where it enters the popliteal vein. It communicates with the long saphenous vein by several small veins (Fig. 14.3).

2. The deep veins

These are intra and inter-muscular veins, which accompany the named arteries within the musculofascial compartments of the lower extremity and are usually given the same name. They often run as paired venae comitantes below the knee.

3. The perforating veins

These perforate the deep fascia to connect the superficial and deep venous systems. The valves in the perforating veins direct blood from the superficial to the deep veins. The skin of the lower part of the leg is drained by special perforators which are either:

- Indirect perforators: pass from the superficial veins to the soleal plexus and then to the deep veins.
- Direct perforators: pass from the posterior arch (branch of the long saphenous vein) to the posterior tibial vein.

The perforators on the medial side of the leg are situated at about 6, 12 and 18 cm above the sole of the foot.

The venous system of the lower limb is characterized by the presence of unidirectional bicuspid valves which allow blood to pass from below upwards and from superficial to deep veins (Fig. 14.4). The venae cavae are exceptions as they have no valves. The valves start to appear in the venules that are about 1 mm in diameter.

Factors that help the venous return from the lower limbs

- The muscle pump which needs strong muscles and intact deep fascia.
- Transmitted arterial pulsations to the venae comitantes.
- The unidirectional valves.
- The negative intrathoracic pressure.

Venous thrombosis-General principles

Types

- Superficial venous thrombosis.
- Deep venous thrombosis occurring mainly in the calf or iliofemoral veins. Less common sites are the inferior vena cava, subclavian, axillary or portal veins.

Pathogenesis

Virchow in 1856 mentioned his triad for the possible factors predisposing to venous thrombosis.

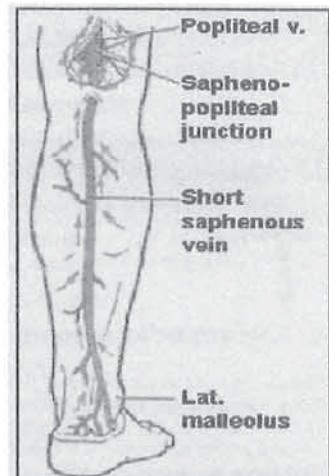


Fig. 14.3. Short saphenous vein.

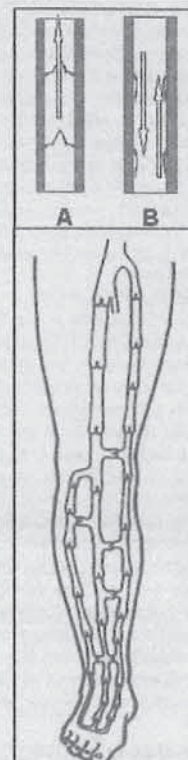
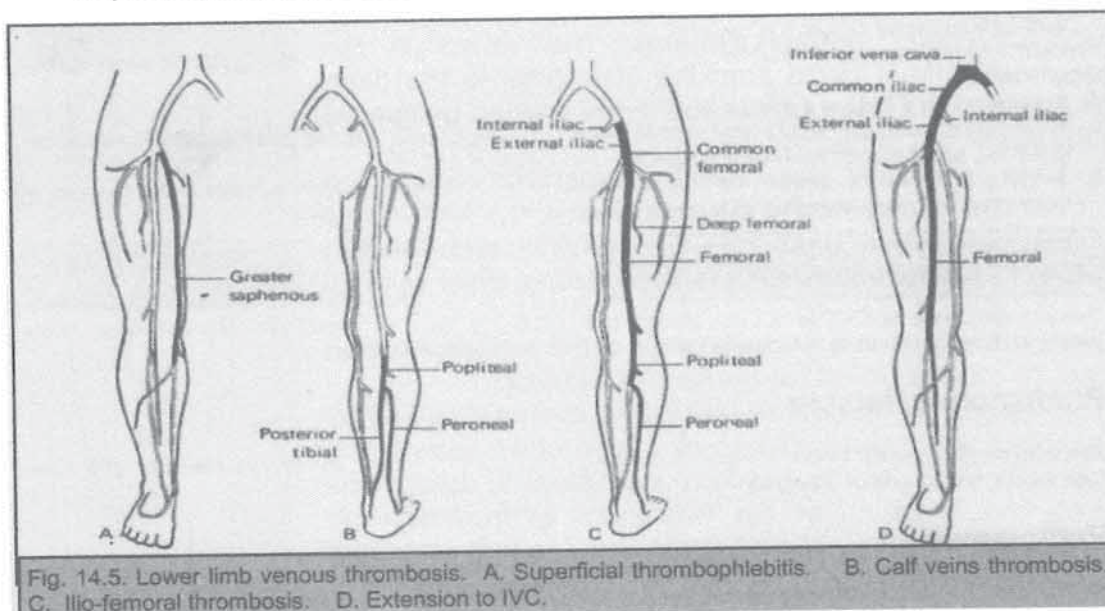


Fig. (14.4) Venous valves
A. Competent; B. Incompetent

1. Damage to the endothelial lining of the vein wall due to
 - Trauma to the vein wall, e.g., during pelvic operations.
 - Inflammatory process near the vein, e.g., pelvic sepsis.
2. Venous stasis due to
 - Prolonged bed confinement, long trips, or casts.
 - Congestive heart failure.
 - Venous compression by tumours, a pregnant uterus or pillows under the knees.
3. Hypercoagulability of blood may be either
 - Primary hypercoagulability due to,
 - Deficiency of antithrombin III, proteins S and C, macroglobulins or antitrypsin.
 - Polycythaemia.
 - Factor V Leiden gene defect or activated protein C resistance.
 - Dysfibrinogenaemia.
 - Secondary hypercoagulability due to oral contraceptives, or malignancy especially ovarian cancer.



Superficial vein thrombosis (superficial thrombophlebitis)

Aetiology

Superficial thrombophlebitis may occur with the following conditions

1. Varicose veins.
2. Veins cannulated for I.V. infusion.
3. After injection of irritant drugs, e.g., diazepam.
4. In association with
 - Buerger's disease (thrombophlebitis migrans).
 - Polycythaemia.
 - Polyarteritis.
 - Visceral cancers, it may be the earliest sign of malignancy (Trousseau' sign).
5. Idiopathic.

Clinical features

The vein becomes red, painful and cord like. There may be slight pyrexia.

Complications

If infection sets in, rapid upward spread occurs with the danger of extension to the deep veins via the communicating veins. In this condition if clinical tenderness is detected less than 10 cm from the saphenofemoral junction, proximal ligation and disconnection should be done.

The thrombus is adherent to the vein wall as there is inflammation. Pulmonary embolism never occurs.

Treatment

1. Compression by elastic stocking or compression bandage.
2. Anti-inflammatory drugs, e.g. aspirin. These are usually enough for treatment of the majority of cases. The following are resorted to under special circumstances.
3. Antibiotics only if there is evidence of infection.
4. Anticoagulant therapy (heparin and warfarin) is given in severe progressive cases (ascending thrombophlebitis), and if associated DVT is suspected.
5. Surgery. Prophylactic sapheno-femoral or sapheno-popliteal disconnection is done if there is tenderness less than 10 cm from the junction.

Deep venous thrombosis (DVT)

The true incidence of DVT is not exactly known as many cases pass unnoticed. The incidence varies greatly in different countries, which may be due to racial and seasonal variations.

Predisposing factors

All the factors mentioned before under Virchow's triad. Added to these are obesity, old age, oral contraceptives intake, previous DVT, malignancy and major trauma.

Pathogenesis

1. The process usually starts in the calf venous sinuses or in the iliac and femoral veins by adherence of platelets to the endothelial surface forming a grey cluster.
2. Then more platelets adhere. Fibrin and RBCs are deposited as layers in-between the platelets giving a laminated appearance known as the lines of Zahn.
3. When the vein is totally occluded, non adherent, jelly-like propagated thrombus spreads up the vessel as far as the next major tributary. This thrombus is dark red and consists only of fibrin and red cells. At this stage the thrombus is loosely attached and it can be easily detached leading to pulmonary embolism.
4. Later, the thrombus becomes tightly adherent to the vein wall by fibrin deposition. It then organizes and contracts thus producing destruction of the valves and luminal narrowing, which are responsible for the eventual development of the post-phlebotic limb syndrome.
5. Later on the processes of fibrinolysis and phagocytosis start and help in recanalization of the vein but the valves are permanently destroyed. 90% of occluded veins will be recanalized within 9 months while 10% of affected veins remain occluded for life.

Clinical features

The clinical picture of DVT varies greatly from silent cases to severe local and systemic symptoms.

The classical picture. There is a triad of pain, tenderness and swelling.

1. **Pain.** There is usually aching discomfort and tightness in the involved calf or thigh, which are aggravated by muscular exercise.
2. **Swelling.** This is the most reliable physical sign. It is evidenced by measuring the difference in the circumference between both sides. In calf thrombosis the swelling is limited to the foot and ankle, in femoral thrombosis the swelling involves the calf and lower part of the thigh, while in ilio-femoral thrombosis there is massive swelling affecting the whole lower limb.
3. **Tenderness** is present on grasping the affected calf or thigh or on compressing the muscles by the two hands against the bones. Dorsiflexion of the foot causes pain in the calf (Homan's sign). However this sign is not reliable.

The complication group

1. **Phlegmasia alba dolens.** Massive iliofemoral DVT may be associated with severe arterial spasm, so the limb becomes pale, white, and massively swollen with absent peripheral pulses.
2. **Phlegmasia cerulea dolens** Massive iliofemoral DVT may be associated with severe congestion and cyanosis and the whole lower limb looks massively swollen and blue and if not properly treated it may lead to venous gangrene.
3. **Venous gangrene.**
4. **Pulmonary embolism** manifested by severe chest pain, dyspnoea and haemoptysis.

The asymptomatic group

Silent DVT is a frequent occurrence. There are no local symptoms and the patient may present later with either pulmonary embolism or with the manifestations of a post-phlebitic limb. However, it may be suspected by the presence of unexplained rise of temperature or pulse rate.

Investigations

As clinical examination is not reliable (incorrect in about 50% of cases), investigations should be done before starting treatment.

1. **Doppler Ultrasound.** Upon applying the ultrasound probe over a patent vein, a continuous sound is heard (venous hum). If the probe is applied over the femoral vein and pressure is applied to the calf, the hum gets accentuated into a roar due to increased blood flow. If there is thrombosis in the popliteal or femoral veins, the roar does not occur. It is a simple and rapid method and is accurate in 80-85% of cases. Ultrasound is insensitive in calf vein thrombosis.
2. **Duplex ultrasound scan.** This method combines Doppler ultrasound flow analysis with ultrasound imaging. Recent instruments employ colour flow imaging which permits determination of flow direction and turbulence and detects partially occlusive thrombi. Its sensitivity and specificity reach 90-100% and most duplex errors are in below knee veins. Therefore, duplex scanning is considered the standard test for diagnosis of DVT. Its accuracy approaches that of venography but is safer and simpler. Table 14.1 illustrates the Doppler and duplex scan findings in cases of DVT.

Table (14.1) Doppler and duplex scan findings in cases of DVT

	Normal veins	DVT
Vein diameter	Normal	Dilated vein
Blood flow	Spontaneous	Poor
Echogenic material in lumen	None	Present
Distal compression	Augments blood flow	Poor augmentation
Blood flow with respiration	Phasic flow with respiration	Loss of phasic flow with respiration

3. **Enhanced helical computed tomography (CT)** can show the thrombus even in small veins.
4. **Magnetic resonance venography** gives excellent delineation especially for the pelvic veins.

¹²⁵I-fibrinogen uptake and ascending venography are no more used for the diagnosis of DVT. They have been replaced by duplex ultrasound.

Differential diagnosis

- Contusion of calf muscles.
- Rupture of plantaris tendon. Both previous conditions occur during exercise and can produce a swollen painful calf which is usually difficult to differentiate from DVT. Duplex scan is needed to establish the diagnosis.
- Calf haematoma.
- Ruptured Baker's cyst.
- Lymphatic obstruction The swelling is chronic and usually non pitting.
- Cellulitis There are systemic symptoms and signs of inflammation and the leg is hot and red.

Prevention of postoperative DVT

Physical measures to reduce venous stasis

1. Early ambulation after operations.
2. Active leg exercises while in bed.
3. Adequate postoperative hydration. The above three measures are routine precautions for all postoperative patients.
4. Elastic stocking support especially in the elderly.
5. Intra-operative intermittent pneumatic external calf compression.

Prophylactic anticoagulants

Indications.

1. High-risk cases for development of DVT
2. History of DVT or pulmonary embolism.
3. Major surgery, particularly cancer operations.
4. Females on contraceptive pills.
5. Elderly patients.
6. Obesity.

Methods

- (a) Low-dose heparin (Mini-Heparin) 5000 IU subcutaneously 2 hours before operation and then every 12 hours until the patient is ambulant (5-7 days) proved very effective in lowering the incidence of DVT by 50%. Prophylactic heparinization should not be used if a large raw area is left after surgery.
- (b) Low molecular weight heparin is gaining popularity because it is given once daily and has lower risk of bleeding, making it more suitable for use at home after discharge.

Treatment

The objectives of treatment are

- Prevention of formation of new thrombi.
- Prevention of pulmonary embolization.
- To minimize venous valves damage.

1. Bed rest and elevation of the lower limb

- The patient should be confined to bed with the feet elevated 15-20 degrees above the level of the heart. Elevation reduces oedema and pain and increases venous return thus preventing further thrombosis.
- Application of an elastic bandage or stocking will also help venous return. Thrombi usually take 7-10 days to become adherent to the vein wall, the patient should be kept in bed for this period. Usually the swelling, pain, and tenderness on grasping have resolved by this time. Graduated ambulation with elastic support is then allowed but standing and sitting with the legs dependent are forbidden because the accompanying rise in venous pressure aggravates oedema and discomfort. These measures are continued for 3 to 6 months until recanalization and collateralization develop.

2. Anticoagulant therapy (Fig. 14.6)

Heparin is an acid mucopolysaccharide.

Mode of action

- Enhances the activity of the naturally occurring antithrombin III. The anti-thrombin III-heparin complex neutralizes the effect of thrombin.
- Neutralizes factors IX, X, and XI.
- It may also have a mild thrombolytic effect as it can cause mild activation of fibrinolysis.
- There are two types of heparin
 - Low molecular weight heparin (fractionated heparin)
 - Tinzaparin 175 IU/Kg/24 hours subcutaneously.
 - Enoxyparin 1 mg/Kg/12 hours subcutaneously.
 - Unfractionated heparin is administered either by
 - Continuous IV infusion A loading dose of 80 IU/Kg then maintenance dose of 18 IU/Kg/hour. This is the ideal method, but it requires hospitalization and a pump which controls the dose per hour.
 - Bolus therapy A loading dose of 80 IU/Kg then maintenance dose of 70 IU/Kg/4 hours.
 - The dose of heparin is monitored by measuring the activated partial thromboplastin time (APTT) which should be kept between 2-2.5 times the control value.

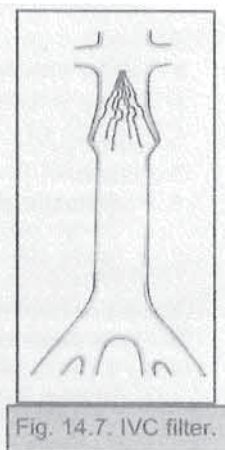
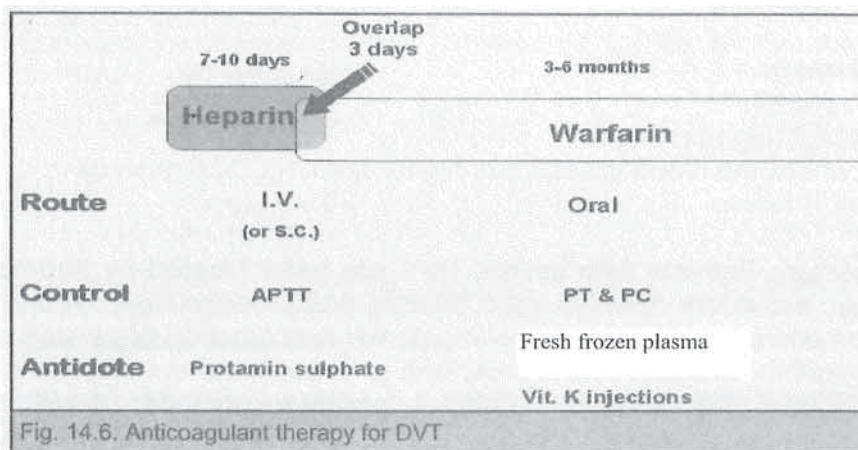
Heparin is given until all signs of active thrombosis subside which is clinically known by disappearance of pain and tenderness on grasping. This usually takes 7-10 days. Table (14.2) illustrates the differences between unfractionated heparin and low molecular weight heparin.

Table (14.2) Differences between unfractionated heparin and low molecular weight heparin

	Unfractionated heparin	Low molecular weight heparin
Mode of action	Antithrombin III	Anti Xa
Half life	1.5-2 hours	12 hours
Route of administration	I.V.	Subcutaneous
Monitoring	Activated partial thromboplastin time	Not required – if needed → level of Xa
Incidence of heparin induced thrombocytopenia	1-5%	<2%
	The patient should be hospitalized	May be taken at home

Complications

1. Bleeding due to over dosage usually manifests by subcutaneous bruises, epistaxis, bleeding gums, hematuria, or gastro-intestinal bleeding. This can be avoided by proper control of the dose by repeated APTT and can be treated by giving protamine sulphate 1 mg IV for every 100 IU of heparin. The use of subcutaneous low molecular weight heparin is less likely to cause bleeding.
2. Failure to respond to heparin (heparin resistance) It may be mild and requires only increasing the dose of heparin.
3. Heparin induced thrombocytopenia occurs usually in the second week of therapy. If the platelet count drops below $100,000/\text{mm}^3$, heparin should be stopped.



Oral anticoagulants (coumarin derivatives). The most commonly used is warfarin.

Mode of action

These drugs block the synthesis of at least 4 vitamin K dependent clotting factors (prothrombin and factors VII, IX and X). For this reason its anticoagulant effect is delayed. Oral anticoagulants inhibit the synthesis of protein C and S which has an anticoagulant effect and so there is a period of relative hypercoagulability during the first few days of warfarin therapy. This period should be covered by continuing heparin therapy together with the warfarin for the first 3 days.

Administration

1. Basal level of prothrombin time and concentration are estimated prior to start of warfarin.
2. An initial dose of 10 mg warfarin is followed by 5 mg daily dose.
3. Discontinue heparin after 3 days of overlap treatment.
4. Five days after the start of warfarin the prothrombin time (PT) and concentration (PC) are measured and the dose is adjusted to reach the therapeutic goal of PC 30-40%.
5. PT and PC should be repeated every 2 weeks during the course of treatment. The dose is adjusted accordingly. These tests are more accurately expressed as the international normalized ratio (INR), which should be to between 2-3 times the control value.
6. Oral anticoagulants are given for 3-6 months which is the time needed for recanalization and collateralization as evidenced by duplex examination. In some patients who are liable to rethrombosis, warfarin is given indefinitely.

Complications

Bleeding is the main problem and is treated by lowering the dose and in severe cases 10-20 mg vitamin K IV injection can be given. In rare cases severe shock may occur from massive retroperitoneal haemorrhage. It is treated by fresh blood or fresh frozen plasma transfusion and vitamin K injection. Interaction may occur between oral anticoagulants and other drugs like aspirin and other non steroidal anti-inflammatory agents, barbiturates or H₂ blockers and thus lead to an increase in the anticoagulant effect.

Contraindications to anticoagulant therapy

1. Absolute

- Trauma to or recent operation on the brain or spinal cord.
- Haemorrhagic diathesis.

2. Relative

- Major visceral injury.
- Major acute fractures.
- History of cerebral haemorrhage.
- Hypertension where the blood pressure is higher than 180/120 mm Hg.
- Peptic ulcer.

3. **Thrombolytic therapy** Patients with severe DVT are better treated by thrombolytic therapy. Fibrinolytic activators dissolve fresh thrombi and produce rapid clearance of the occluded veins and may preserve the competence and function of venous valves better than anticoagulant therapy. They, thus, limit or prevent the development of the postphlebotic syndrome. Examples of fibrinolytic activators include; streptokinase, urokinase and tissue plasminogen activator (TPA). The last drug is prepared by recombinant gene technology. The effect of these drugs is at its best if they are given in the first 3 days of thrombosis, after that they have no advantage over heparin.

Complications

1. Allergic reactions. It is better to give corticosteroids prior to thrombolytic drugs.
2. Severe bleeding. These drugs are contraindicated in old age, hypertension, peptic ulcer and a history of haemorrhagic diathesis.
3. They are very expensive.

4. Insertion of vena caval filter is indicated in

- Progressive thrombosis or recurrent pulmonary embolism in spite of anticoagulant therapy.
- If there is a contraindication to the use of anticoagulants.

Procedure

Under local anesthesia and radiographic control the Greenfield filter is placed by the percutaneous or open exposure method through the jugular vein. The filter is usually placed in an infrarenal position (opposite the second lumbar vertebra). Complications of the procedure include hemorrhage, injury of IVC, perforation to the aorta, migration and recurrent emboli in less than 5% (Fig. 14.7).

Axillary-Subclavian DVT

Incidence 2-3% of all cases of DVT.

Aetiology

1. **Spontaneous (primary).** This is usually preceded by unusual muscle activity of the upper extremity (sports, or occupational) due to compression of the vein in the costoclavicular space.
2. Secondary due to
 - a. Central venous catheters.
 - b. Metastatic tumours in the axilla.

- c. IV chemotherapy or parenteral hyperalimentation given through upper limb veins.

Clinical features

- Pain and swelling of the entire upper extremity.
- Fullness of the infraclavicular fossa.
- Prominent venous collaterals over the shoulder and anterior chest wall.
- The limb may be cyanosed.

Treatment is similar to that of lower limb DVT. Resection of the first rib or scalenus anterior may be required if the vein is compressed at the thoracic outlet.

Pulmonary embolism

Between 2-3% of all hospital mortalities are due, wholly or in part, to pulmonary embolism. Pulmonary emboli originate from thrombi in the venous circulation. They are, therefore, referred to as thromboemboli. Fragments of these thrombi are dislodged and float to the right side of the heart which pumps them into the pulmonary artery and get impacted in the pulmonary artery or one of its branches.

Aetiology

All the predisposing factors which lead to DVT are important in the aetiology of PE.

Pathophysiological sequelae

The effects of PE are relatively insignificant until more than 25% of the pulmonary artery circulation is occluded. Cardiac output remains normal until pulmonary artery occlusion exceeds 50% (massive embolism). Underlying cardiac or respiratory insufficiency contributes significantly to premature failure of cardiac output.

- Decreased pulmonary blood flow which results in
 - Reduced cardiac output (CO) leading to systemic hypotension and shock.
 - High ventilation perfusion ratio due to reduced pulmonary arteriolar circulation in areas of normal ventilation.
 - Myocardial hypoxia.
- Pulmonary infarction occurs in less than 10% of PE cases. The development of an infarction depends on completeness of occlusion and effectiveness of collateral circulation.

Clinical features

The clinical picture of pulmonary embolism is determined by the site where the embolus impacts. Accordingly three types of embolism are recognized

1. Small emboli are impacted in the peripheral arterioles. They are usually silent. Recurrent small emboli will lead to pulmonary hypertension.
2. Medium sized emboli lodge in the branches of the pulmonary arteries and result in pulmonary infarction. This leads to the classical picture of severe pleuritic pain, dyspnoea and haemoptysis. This classic picture is not common.
3. Large emboli are impacted either in the main pulmonary artery itself or in one of its major branches leading to massive pulmonary embolism. There is severe precordial pain, tightness in the chest, marked dyspnoea, severe hypotension and marked tachycardia. Sudden death may occur.

N.B.: It should be stressed that in many patients with pulmonary embolism, there is no clinical evidence of deep vein thrombosis in the lower limbs.

Unexplained dyspnoea or heart failure appearing in a hospitalized patient is very suggestive of pulmonary embolism.

Table (14.3) illustrates the frequency of symptoms or signs of pulmonary embolism.

Table (14.3) Frequency of symptoms and signs of pulmonary embolism

Dyspnoea	100%	Rales	50%	Haemoptysis	25%
Tachypnoea >20 min	95%	Cough	50%	Cyanosis	15%
Tachycardia >90/min	70%	Pleuritic pain	35%	Syncope	5%
Fever	55%	Clinical DVT	25%		

Differential diagnosis

- Pneumonia.
- Congestive heart failure.
- Myocardial infarction.
- ARDS.

Investigations

1. **D-dimer.** This is a degradation product of fibrin. It is elevated with DVT and PE. A normal value excludes pulmonary embolism.
2. **Blood gases.** The most consistent finding is low PaO₂. PCO₂ remains normal.
3. **ECG.** The major value is to differentiate PE from myocardial infarction. In 40% of patients with PE there are large P waves, right axis deviation and T wave inversion.
4. **Chest X-ray.** This is entirely normal in 50% of cases or may show are following:
 - a. Prominent pulmonary artery (hilar shadow).
 - b. Diminished vascular markings in the area supplied by the obstructed pulmonary artery.
 - c. Enlarged right ventricle.
 - d. Small pleural effusion.
5. **Duplex scan** of lower limb veins in order to detect occult venous thrombosis.
6. **Ventilation perfusion** pulmonary isotope scan shows lung areas that are ventilated but not perfused.
7. **Pulmonary arteriography.** This is a definitive diagnostic investigation in suspected cases. A venous catheter is passed into the right side of the heart, then the pulmonary artery, to inject a contrast material. Visualization of an intraluminal filling defect or abrupt vessel cut-off or loss of a side branch is diagnostic. Pulmonary angiography though diagnostic is dangerous in shocked patients. Also microemboli might be overlooked with pulmonary angiography.
8. **CT pulmonary angiography** is the most popular imaging study. This is because it is a good diagnostic tool and is non-invasive as there is no need for vascular catheterization.

Treatment

Oxygen inhalation is started on suspicion of pulmonary embolism.

1. **Minor and moderate pulmonary emboli** are treated by anticoagulants.
2. **Patients with massive pulmonary embolism** represent a very serious surgical emergency. Many of these patients are dead before any help can be offered. If the patient could withstand the major embolus, he should be immediately transferred to a cardiothoracic center where urgent pulmonary arteriography is performed (conventional or CT). If the diagnosis is confirmed the following measures are performed
 - a. If a venous catheter was used for angiography it is to be left in the pulmonary artery and 600,000 units of streptokinase are injected followed by 100,000 units/hour for 72 hours. The cardiac function should improve within 6 hours in favourable cases.
 - b. If deterioration sets in, urgent pulmonary embolectomy is performed.
3. **Vena caval interruption** (Fig. 14.7). The objective is immediate prevention of recurrent PE.

Indications

- When anticoagulant therapy is contraindicated because of major bleeding complications.
- Recurrent embolism in spite of anticoagulant therapy.
- First PE in high risk patients.
- Following pulmonary embolism.

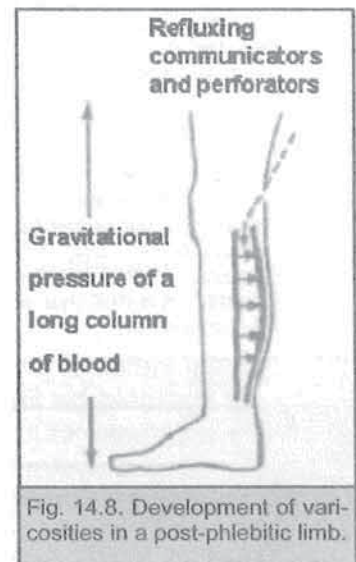


Fig. 14.8. Development of varicose veins in a post-phlebotic limb.

Varicose veins of lower limbs

This is a frequently occurring condition in which the superficial veins are dilated, elongated and tortuous.

Types

There are two types of the disease

1. **Primary varicose veins** This means that the cause is not known.

Theories of aetiology

There are two theories, neither of which satisfactorily explains all cases.

- a. The weak wall theory assumes an inherited weakness of the vein wall, producing venous dilatation even with normal pressures. Secondary valvular incompetence will occur.
- b. The congenital valvular incompetence theory postulates that the fundamental abnormality is sequential incompetence of valves either in the main saphenous trunks or in the communicating veins.

A positive family history can only be found in 50% of cases of primary varicose veins.

Aggravating factors

1. Female sex. The condition is commoner in females.
2. High parity.



Fig. 14.9. Disfigurement that is caused by varicose veins.

3. Occupations requiring prolonged standing.
4. Marked obesity.
5. Constricting clothes.
6. Oestrogens intake, e.g. contraceptive pills.

2. Secondary varicose veins. These are varicose veins that develop secondary to one of the following

- a. Deep venous thrombosis. This is the usual cause of secondary varicose veins. Recanalization of the thrombosed deep veins leaves the valves of the perforating veins incompetent leading to reflux of blood (Fig. 14.8). This places an unusual strain on the superficial veins which have little external support, so they progressively dilate.
- b. Arterio-venous fistula. Varicose veins are prominent with traumatic or congenital (Klippel-Trenaunay syndrome) A-V fistulae. Here, the arterial pressure is transmitted to the deep system leading to valvular incompetence and reflux of blood to the superficial veins which dilate and become varicose.
- c. Pelvic tumours and pregnancy. Due to compression of the pelvic veins, the pressure in the deep venous system of the lower limbs is elevated, leading to reflux of blood to the superficial system and valvular incompetence.

It should be firmly realized that the presence of secondary varicose veins is not a disease of its own. It is actually one of the manifestations of a disorder of deep venous system, usually following deep vein thrombosis, hence the name post-phlebitic syndrome.

Clinical features

Symptoms

1. Cosmetic disfigurement (Fig. 14.9).
2. Aching, and discomfort of the limb and restless leg usually described as dull, heavy, bursting sensation with sense of hotness usually at the end of the day or on prolonged standing and is relieved by elevating the limbs.
3. Night cramps.
4. Mild swelling occurs at the end of the day, particularly with secondary varicose veins.
5. Pigmentation. This is more in secondary varicose veins due to ooze of blood in the subcutaneous tissue and deposition of haemosiderin.
6. Itching.
7. Ulceration is more with secondary varicose veins.

Signs

The aim of examination in a patient with varicose veins is to get the following conclusions



1. The anatomical distribution of the veins.
2. Are the varicosities primary or secondary?
3. The competency of the saphenofemoral junction and other communicating veins.
4. The condition of the deep system of veins.
5. The presence of complications.

The patient should be examined while standing and should be exposed up to the umbilicus. The following signs are to be noticed

1. Varicose veins appear as elongated, dilated and tortuous veins.
2. The shape of varicose veins may be tubular, saccular, serpentine or spider.

3. The veins may belong to the long or short saphenous system in case of primary varicose veins or may be arranged haphazardly in case of secondary varicose veins.
4. Presence of veins crossing the suprapubic regions denotes secondary varicose veins caused by narrowing of the external iliac vein.
5. Presence of complications as oedema, eczema, liposclerosis or ulceration is in favour of secondary varicose veins. Table 14.3 illustrates the differences between primary and secondary varicose veins.
6. Palpation of a thrill over the veins denotes the existence of an arterio-venous fistula.
7. Palpation of a thrill over the saphenofemoral junction on cough denotes an incompetent saphenofemoral junction.

Table (14.3) Differences between primary and secondary varicose veins

	Primary V V		Secondary V V	
	Idiopathic	Aetiology	Secondary to previous DVT, A-V fistula, pelvic tumours or pregnancy	
	Slight or absent	Pain	Marked	
	Along the long or short saphenous veins	Distribution	Haphazard Veins crossing the groin may be seen	
	Minimal or absent	Complications	Oedema pigmentation, eczema, and ulceration are frequent features	

Special tests

1. Trendelenburg's test

Aim

- a. Detection of sapheno-femoral incompetence.
- b. Detection of incompetence of communicating veins.

Method

- a. The patient lies supine and raises his leg to empty the veins.
- b. A venous tourniquet is applied just below the saphenous opening.
- c. The patient is then asked to stand up.
- d. The tourniquet is then released.

Results

- a. If the sapheno-femoral valve is normal, the veins will fill slowly from below upwards.
- b. If the sapheno-femoral valve is incompetent, the veins will fill rapidly from above downwards once the pressure on the valve is released.
- c. If the veins fill rapidly while the tourniquet is still in place, it means incompetent communicating veins. In this case the multiple tourniquet test is done to identify the site of the incompetent communicating veins.

2. Multiple tourniquet test

Aim Localizing incompetent communicating veins when Trendelenberg's test shows rapid filling of varicosities while the sapheno-femoral junction is still occluded.

Method: The steps are similar to those of the Trendelenburg's test but with the application of three tourniquets; one just below the knee, the second at the junction of the lower third of the thigh with the upper two thirds, and the third tourniquet at the junction of the lower two thirds of the thigh with the upper third. The patient is then asked to stand up and the tourniquets are released one by one from below upward.

Result: Rapid filling in any segment indicates the site of regurgitation.

Perthe's test

Aim Detection of the patency of the deep venous system.

Method

- a. The patient lies on his back and the lower limb is elevated.
- b. An elastic bandage is applied firmly from the toes to the upper third of the thigh.
- c. The patient is then asked to stand and walk in situ for 5 minutes.

Result If the deep system is blocked, the patient will complain of pain in the leg and inability to continue the exercise.

Modified Perthe's test

Aim Detection of the patency of the deep system.

Method While the patient is standing, a tourniquet is applied just below the sapheno-femoral junction and the patient is asked to walk quickly in situ for five minutes.

Result If the varicose veins disappear, this means that the deep system is patent and competent as the efficient leg muscle pump creates a negative pressure in the deep veins, thus sucking the blood from the superficial venous system. If the veins become more engorged, then the deep veins are obliterated or incompetent.

Complications

Complications are more frequent in association with secondary varicose veins. These are mainly manifestations of the post-phlebitic syndrome. The varicose veins themselves tend to produce no or minimal complications.

1. Oedema.
2. Subcutaneous bruises due to rupture of small veins as a result of venous hypertension.
3. Itching, dermatitis and eczema are due to deposition of haemosiderin in the subcutaneous tissues.
4. Liposclerosis. The extravasated fibrinogen leads to fibrous tissue formation. The soft supple subcutaneous fat is replaced by tough fibrous tissue.
5. Recurrent superficial thrombophlebitis.
6. Venous ulceration.
7. The combination of the above mentioned complications, together with chronic leg pain, constitute the "post-phlebitic" syndrome as they usually follow previous DVT with incompetent perforating veins.
8. Haemorrhage may occur due to rupture of a varicose vein especially when the overlying skin is thin. It can be initially stopped by elevation and compressor bandage. Later treatment is by injection sclerotherapy.

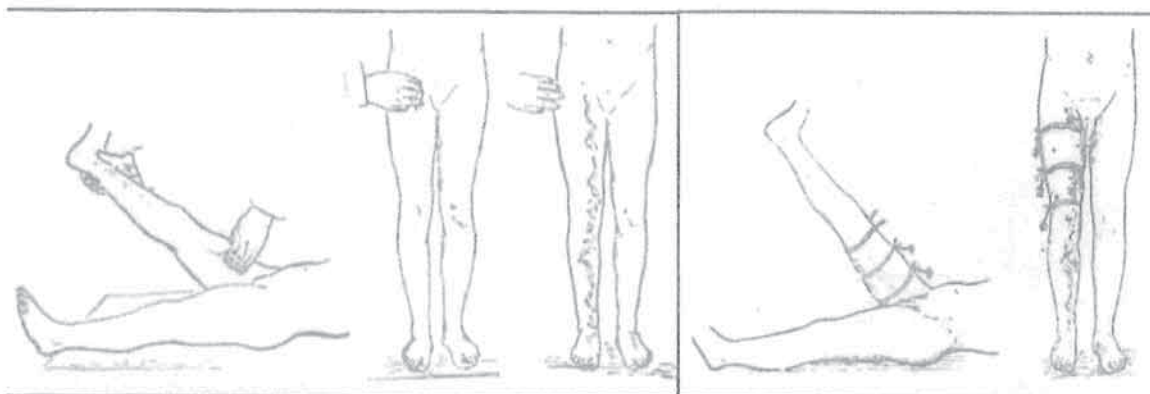


Fig. (14..10) Trendelenberg's test. In this cases it shows incompetent sapheno-femoral junction

Fig. (14..11) Multiple tourniquet test

Investigations

The aim is detection of the sites of incompetent communicating veins and to verify the patency of the deep veins. It should be noted that most cases of primary varicose veins require no investigations because careful clinical assessment provides enough information.

Duplex ultrasound imaging has largely replaced venography as the investigation of choice. It serves accurate detection of incompetent perforators, and detection of accompanying deep vein thrombosis in suspected cases.

Treatment

Primary varicose veins

1. **Conservative treatment** Patients with minor cosmetic or spider varicosities are treated by reassurance and elastic stockings.
2. **Injection sclerotherapy**

Indications

1. Small unsightly veins.
2. Localized dilated superficial veins.
3. Lower leg perforators.
4. Recurrent or persistent veins after operation.

Principle

Sclerosants like 5% ethanolamine oleate, or 3% sodium tetradecyl sulphate should be injected in an empty vein to induce injury of the endothelial layer of intima. If compression is then applied, the two walls of the vein will adhere together without any intervening blood clot thus permanent occlusion of the vein occurs. If injection is done with the vein full of blood, thrombosis will occur which later recanalizes with recurrence of varicosities.

Technique

1. A venous tourniquet is applied over the thigh while the patient is standing and the site of the vein is identified and marked on the skin by a skin marker.
2. The patient is asked to lie down and a thin hypodermic needle is inserted into the vein.
3. The limb is then elevated by an assistant and one ml of the sclerosant material is injected.
4. An elastic bandage is then applied over the whole leg from the toes to the knee and is left for 4-6 weeks.

5. Immediately after the application of the bandage, the patient is instructed to walk for a long distance in order to flush any amount of sclerosant that might have reached the deep veins.

Complications

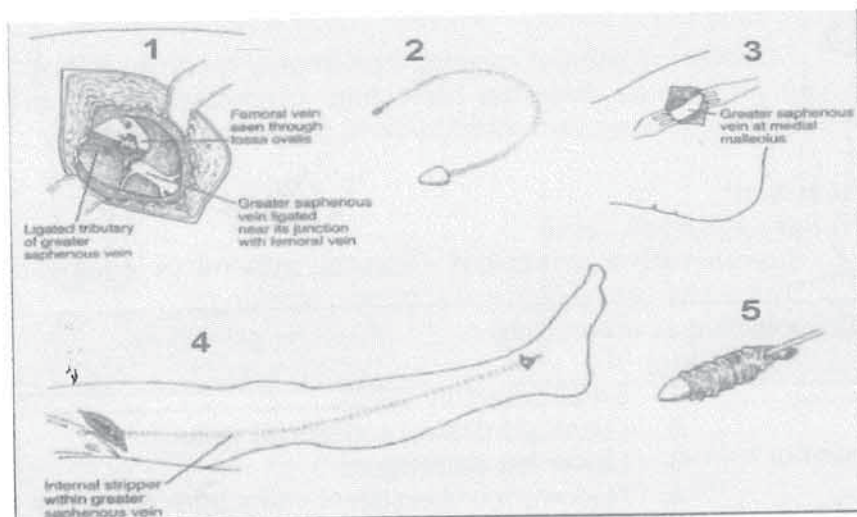
1. Extravasation of sclerosant material under the skin leads to sloughing of the skin and poor cosmetic results.
2. Deep venous thrombosis may occur if a large amount of the sclerosant material reaches the deep system undiluted so no more than 1 ml is injected at any point and no injection is done for veins above the knee.

3. Surgery

Patients with clear evidence of long or short saphenous incompetence or a combination of the two should be treated by sapheno-femoral or saphenopopliteal disconnection combined with stripping (Fig. 14.12). Ligation of the saphenofemoral junction is called Trendelenburg's operation. The saphenous vein should be disconnected flush with the femoral or popliteal vein and all tributaries near their termination must be ligated and divided to prevent recurrence. Then the long saphenous vein is stripped from above the knee to the groin to avoid injury of the saphenous nerve. The short saphenous is stripped from the lateral malleolus to the popliteal region.

Fig. (14.12) Surgery for long saphenous primary varicose veins.

1. Ligation of sapheno-femoral junction and ligation of tributaries.
2. Stripper.
3. Exposure of long saphenous in front of the medial malleolus, or better above the knee
4. Stripper is passed through the whole length of long saphenous vein.
5. Stripped out long saphenous vein.



Secondary varicose veins

1. Post phlebitic (i.e. following deep venous thrombosis). The majority of cases are treated conservatively by elastic stockings. Rarely the varicosities are large enough to require active treatment. In these cases verify that the deep system is recanalized clinically and by duplex, then treat as primary varicose veins.
2. Varicose veins complicating AV fistula. Surgical treatment of the fistula is usually followed by marked regression of varicosities. If residual veins remain, treat as primary varicose veins.
3. Varicose veins occurring during pregnancy. A complete elastic stocking from the toes up to the groin is applied all through the period of pregnancy. After labour any residual veins are treated as primary varicose veins.

Venous ulcers

Venous ulcers constitute 90% of chronic leg ulcers.

Aetiology

1. Venous ulcers usually occur in a post-phlebotic limb due to previous deep vein thrombosis,
2. A much less common cause is arterio-venous fistulas.
3. Rarely the ulcer is a complication of primary varicose veins. In this case it is usually transient and heals rapidly.

Pathogenesis

1. Recanalization of DVT is commonly followed by dysfunction of the valves of both the deep veins and the perforating veins. Furthermore it may produce narrowing of a large deep vein.
2. As the skin of the lower part of the leg is drained directly by the perforators which drain into the deep system, it follows that incompetence of these perforators leads to high venous pressure in the skin particularly that lying on the medial side of the lower third of the leg (ulcer bearing area).
3. The combination of venous hypertension, eczema and liposclerosis (fibrosis of subcutaneous fat) will eventually lead to ulceration after minor trauma.
4. Local hypoxia due to venous stasis and impaired nutrition lead to the liberation of free oxygen radicals which are toxic to the tissues.

Clinical features

The clinical features of a venous ulcer are characteristic.

1. It is usually situated in the ulcer bearing area or around the medial malleolus (Fig. 14.13).
2. The skin surrounding the ulcer shows eczema, itching marks and induration.
3. Secondary varicose veins may be present.
4. The ulcer takes a long time to heal and is liable to break down after healing.

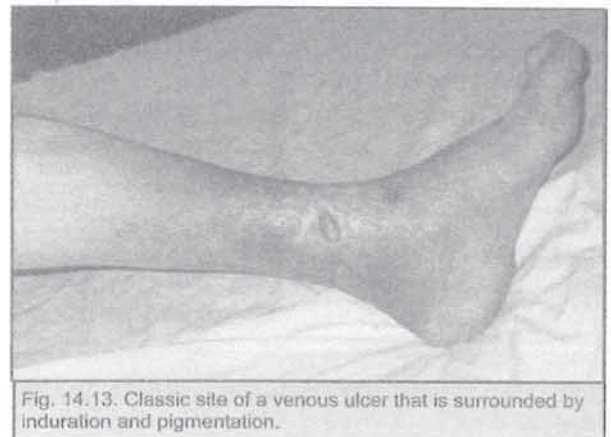


Fig. 14.13. Classic site of a venous ulcer that is surrounded by induration and pigmentation.

Treatment

1. **Conservative treatment is indicated** for all cases.
 - a. Elevation of the foot in bed.
 - b. Ulcer care Moist saline dressings twice weekly. Topical antibiotics should not be used as they may aggravate the condition by inducing an allergic reaction.
 - c. Elastic stocking or crepe bandage. Compression is the most important item in conservative treatment.
 - d. Some medications are said to accelerate ulcer healing
 - i. Pentoxifylline (Trental)
 - ii. Prostaglandin E1 analogue
 - iii. Diosmin

Conservative treatment is usually successful in allowing the ulcer to heal within a few weeks. The problem is that, once the patients return to normal activity, most ulcers will recur.

If there is evidence of incompetent perforators in the leg, they can be either injected or excised.

N.B. Previous operations as Cockett's or endoscopic subfacial ligation of perforators are not commonly performed nowadays.

LYMPHATIC SYSTEM

Anatomy

Lymphatic vessels

Thoracic duct

Formation. All lymph from the lower limbs, pelvis, abdominal viscera and parietes is ultimately drained by three lymph trunks, two lumbar and one intestinal, which unite in the upper part of the abdomen to form the cisterna chyli.

Course. This passes upwards and narrows a little, into the posterior mediastinum and is known as the thoracic duct. This duct receives many lymphatics inside the chest and then it ascends in the left supraclavicular region.

End. It pours its contents into a large vein; the left subclavian, the left internal jugular or the innominate. The right side of the head and neck is drained by a right jugular lymph trunk, which pours into the right subclavian or innominate vein.

Flow. As with veins, active or passive contraction of skeletal muscles plays an important role in the movement of lymph. The lymphatic valves determine the direction of flow.

Lymphatic vessels in a limb

1. **Superficial lymphatics** (epifascial compartment). These drain the skin and subcutaneous tissues. There are superficial and deep lymphatic plexuses in the dermis of the skin. Eventually lymph passes to larger valved trunks that lie in the superficial fascial (subcutaneous fat) and accompany the superficial veins to reach the draining lymph nodes.
2. **Deep lymphatic vessels.** These are much less in number than the superficial lymphatics. They drain all the tissues within the musculofascial compartment and accompany the deep blood vessels.

Superficial lymphatic vessels of the lower limb pass to the vertical group of superficial inguinal lymph nodes while deep lymphatic vessels pass to the popliteal and then to the deep inguinal lymph nodes.

Lymph nodes

Lymph nodes are periodically interposed throughout the course of the collecting lymphatic channels. Each node has several afferent channels entering through the capsule. Lymph enters the lymph sinuses, bathes the cortical and medullary areas, and exits by a single efferent channel. Normal lymph node architecture consists of cortical and

CHAPTER CONTENTS

- Anatomy
 - Lymphatic vessels
 - Lymph nodes
- Physiology
- Visualization of lymphatics
- Lymphadenopathy
- Inflammatory disorders
 - Acute lymphangitis
 - Acute lymphadenitis
 - Chronic non-specific lymphadenitis
 - Tuberculous lymphadenitis
- Lymphoedema
- Neoplastic disorders
 - Hodgkin's lymphoma
 - Non-Hodgkin's lymphoma
 - Burkitt's lymphoma
 - Leukaemias
 - Secondary deposits

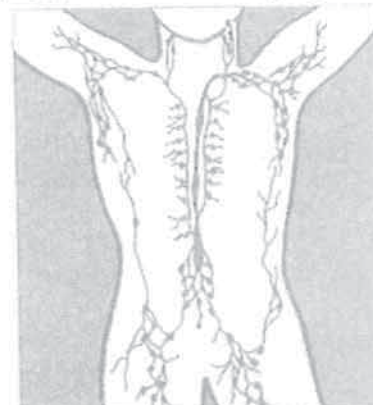


Fig. 15.1. Main lymphatics and lymph node groups.

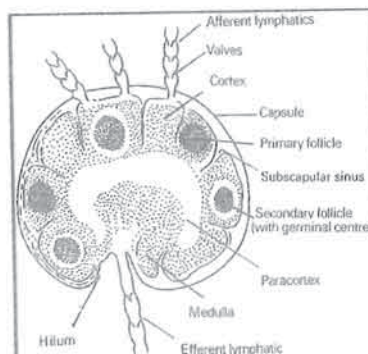


Fig. 15.2. Lymph node structure.

medullary regions. The cortical areas contain predominantly lymphocytes that are arranged in follicles separated by trabecular extensions of the capsule. Within the follicles are discrete germinal centers. The medulla may contain macrophages and plasma cells as well as lymphocytes (Fig. 15.2).

1. **Lymph nodes of the neck.** There are two main chains.

- a. The circular chain: This is actually composed of two circular chains lying horizontally in the neck, an inner and an outer one. The inner chain is formed, inside the mouth, of the two pharyngeal tonsils on either side, the lymphoid tissue on the back of the tongue anteriorly, and the lymphoid tissue on the wall of the pharynx (adenoid) posteriorly. The outer chain lies in the neck around the mandible. It is composed of the submental, the submandibular, the parotid, the preauricular, the postauricular and the occipital lymph nodes. This chain is present on either side of the neck.
- b. The vertical chain: This lies in the neck proper and consists of superficial and deep lymph nodes. The superficial nodes lie in the subcutaneous tissue superficial to the deep fascia. They are scattered irregularly and are surgically unimportant. The deep nodes i.e. deep to the deep fascia, and consist of a median and a lateral group.
 - i. Median along the middle line, from above downwards consists of the prelaryngeal, and the pretracheal (suprasternal) lymph nodes.
 - ii. Lateral, along either side of the middle line of the neck. These are further divided into lower and upper groups by the omohyoid muscle, and into anterior and posterior by the internal jugular vein (not by the sternomastoid muscle).

2. **Axillary lymph nodes.** The axilla is roughly a pyramid with four walls (medial, lateral, anterior and posterior), an apex and a base. The pyramid is obliquely placed so that the apex points upwards and medially and is continuous with the cervicoaxillary canal. The lymph nodes are arranged on the walls, apex and base.

- a. The medial and anterior groups are considered together, and lie behind lower the border of the pectoralis minor muscle and so called the pectoral group.
- b. The lateral group lies on the axillary vessels and brachial plexus and so called the humeral group.
- c. The posterior group lies on the anterior surface of the subscapsularis muscle and so called the subscapsular group.
- d. The central group lies in the base or floor of the axilla.
- e. The apical group lies in the infraclavicular fossa (which is the apex of the axilla).

3. **Inguinal lymph nodes.** These are arranged into 2 main groups.

- a. The superficial inguinal group. These lymph nodes are arranged around the termination of the long saphenous vein superficial to the deep fascia of the thigh. It can be further divided into two chains
 - i. The horizontal chain lies below and parallel to the inguinal ligament. In children some nodes may be found above the inguinal ligament.
 - ii. The vertical chain lies vertically on either side of the upper part of the saphenous vein.
- b. The deep inguinal group. These nodes lie around the upper part of the femoral vein in the femoral canal.

4. **Lymph nodes of the abdomen and pelvis.** These nodes are arranged into two main groups
 - a. Parietal lying behind the peritoneum and in relation to the large blood vessels. These are further divided into
 - i. External iliac nodes.
 - ii. Internal iliac nodes.
 - iii. Common iliac nodes.
 - iv. Sacral nodes.
 - v. Lumbar para-aortic nodes which lie in front, behind and on either side of the aorta.
 - b. Visceral lying alongside the visceral vessels. These are further divided into many groups e.g. coeliac, gastric, hepatic, pancreatic, superior mesenteric, inferior mesenteric, vesical, rectal. ..etc.
5. **Lymph nodes of the thorax.** These are arranged into two groups.
 - a. Parietal lying in relation to the thoracic walls. They can be further differentiated into four groups
 - i. Anterior (internal mammary nodes). These lie alongside the internal mammary vessels. Two or three nodes are usually found in the first and second intercostal spaces.
 - ii. Posterior (posterior mediastinal nodes). These lie behind the pericardium in relation to the oesophagus and aorta.
 - iii. Lateral (intercostal nodes). These lie in front of the necks of ribs or in the posterior parts of the intercostal spaces.
 - iv. Inferior (Diaphragmatic nodes). These lie on the upper surface of the diaphragm.
 - b. Visceral lying in relation to the thoracic viscera. They include
 - i. Superior mediastinal nodes. These lie in front of the trachea, behind the aortic arch.
 - ii. Peritracheobronchial nodes. These comprise many nodes which lie on either side of the trachea and bronchi and in between the two bronchi (interbronchial nodes).

Physiology

The lymphatic system has two main functions

1. **Uptake, transportation, and return of fluid,** macromolecules and foreign substances from the interstitial space to the systemic circulation. It also helps in the clearing of debris from areas of tissue injuries. The amount of lymph returned to the circulation through the thoracic duct in 24 hours approximates the plasma volume. Lymphocytes and other cells enter the central circulation by its channels along with proteins and other large molecules. Chylomicrons and lipoprotein complexes also enter the blood stream via the lymphatic system. In a 70 kg man at bed rest, the thoracic duct lymph flow approximates 2 liters daily with an average protein concentration of 4.8gm per 100 ml.
2. Protection of the host by providing a filtration system that resists infection and impedes the spread of neoplastic diseases. There are three types of lymphocytes. T-lymphocytes are derived from the thymus and mediate cellular immunity. B-lymphocytes are generated independently of the thymus, they produce antibodies mediating humoral immunity. A third type of heterogeneous lymphocytes is known as null or non-B non-T type. The latter are sources of extramedullary haemopoiesis

and can also form specialized cytotoxic lymphocytes that kill target cells in either the presence or the absence of specific antibodies. The different types of lymphatic cells are now identified largely by surface markers.

Visualization of lymphatics

Visual lymphangiography by injection of patent blue violet is performed for

1. Visualization of the lymphatics of a limb in cases of lymphoedema to determine the condition of the lymphatic trunks and the level of lymphatic obstruction.
2. Visualization of the sentinel lymph node mainly in cases of breast cancer.

Isotope lymphography (lymphoscintigraphy)

^{99m}Tc labelled colloidal particles with antimony sulphide are injected in the first web, and the level of radioactivity is measured. It is then measured again after 45 and 90 minutes. The rate of disappearance of radioactivity is estimated. Normally, after 90 minutes, 40% of radioactivity at the injection site should have disappeared. After sometime, radioactivity over the inguinal and pelvic regions is estimated. A curve is drawn showing the relation between the level of radioactivity and time after injection. Normally there is step like rise of activity time curve at the inguinal region. Delineation of the lymphatic tree and lymph flow can be obtained.

Lymphadenopathy

Lymphadenopathy = diseased lymph nodes. The term is used to refer to lymph node enlargement. The causes are

1. **Inflammatory**
 - a. Acute
 - i. Non-specific.
 - ii. Specific. Infectious mononucleosis.
 - b. Chronic
 - i. Non-specific.
 - ii. Specific. Tuberculosis and syphilis.
2. Lymphomas
 - a. Hodgkin's lymphoma.
 - b. Non Hodgkin's lymphoma.
3. Blood diseases
 - a. Acute leukaemia.
 - b. Chronic myeloid leukaemia.
 - c. Chronic lymphatic leukaemia,
4. Lipoidosis.
 - a. Gaucher's disease.
 - b. Niemann-Pick's disease.
 - c. Hand-Schuler-Christian's disease
5. Metastases.

Inflammatory disorders

Inflammation of lymphatic vessels is termed lymphangitis, while that of lymph nodes is called lymphadenitis.

Acute lymphangitis

Lymphatics draining an inflamed area or a septic wound frequently get inflamed, and appear as streaks travelling towards regional lymph nodes. The commonest

organisms are streptococci, but other organisms can give rise to lymphangitis. The end result may be complete obliteration of the affected lymphatics, and if the condition is repeated or involves a major number of lymphatics draining an organ or a limb, permanent oedema or even elephantiasis may be established.

Clinical Picture

Fever, rigors and general constitutional disturbances may be severe. Locally, there is pain, oedema and red tender streaks. The regional lymph nodes are enlarged and tender. The original cause may still exist as an infected wound, or it may have healed and even forgotten by the patient.

Treatment

1. Proper treatment of the cause.
2. Antibiotics, especially penicillin and broad spectrum antibiotics.
3. Local rest of the affected part, and local heat to help resolution.
4. If suppuration occurs, it needs an incision.

Acute lymphadenitis

Infection is carried to lymph nodes along the lymphatics from an inflamed focus.

Pathology

Lymph nodes are enlarged, congested, oedematous, and the cut section shows infiltration with leucocytes. Periadenitis may occur.

Complications

1. Spread to more proximal lymph nodes.
2. Spread to nearby tissues.
3. Suppuration (abscess formation).

Clinical picture

1. The picture of the causative lesion.
2. General constitutional manifestations as fever, rigors and headache.
3. Locally the nodes are enlarged, red, hot, tender, firm or soft, and if suppuration occurs fluctuation will be evident. The intervening lymphatics between the causative focus and affected lymph nodes may be seen as red streaks which are tender (lymphangitis).

Treatment

Treatment of the causative focus may cure the inflamed nodes. General rest and antibiotics, are advised and local heat is applied. If an abscess forms, an incision is required.

Chronic non-specific lymphadenitis

This is very common and clinically an unimportant condition. It is due to chronic infection of a nearby focus e.g. chronic tonsillitis and chronic septic teeth, or is due to incomplete resolution of acute lymphadenitis.

Clinically

The nodes are slightly enlarged, mobile, slightly tender and firm or elastic in consistency. Common examples are:

1. Chronic nonspecific lymphadenitis of the upper deep cervical (jugulo-digastric) nodes in patients with chronic tonsillitis, adenoiditis, or sinusitis.

2. Chronic nonspecific inguinal lymphadenitis in people who walk bare footed, and are, thus, exposed to repeated minor trauma and infection.
3. Chronic nonspecific posterior triangle lymphadenitis is frequently found in children who are infested with headlice (pediculosis).

Treatment

This is directed to the original focus. The nodes need no treatment.

Tuberculous lymphadenitis

There are two main pathological types; the lymph borne, and the blood borne varieties.

Lymph-borne (fibrocasseous) type

This type is common in young patients.

Sites

- The commonest groups of lymph nodes to be affected, are the cervical. The organisms reach them from the tonsils where they are filtered from milk.
- Mediastinal and the axillary groups are also affected especially in children and may or may not be associated with a tuberculous lesion in the lung.
- Abdominal nodes, are commonly affected in children and adolescents, the organisms from ingested milk pass through the lacteals to reach the lymph nodes without affecting the wall of the intestine. In people above the age of 50 years a common finding in the X-ray is multiple mottled calcific shadows of mesenteric glands. These are considered to be old tuberculous nodes that have healed by fibrosis and calcification (tabes mesenterica).

Pathology

The organisms reach the nodes by afferent lymphatics, thus first reaching the capsule and causing tuberculous periadenitis, which causes matting of the nodes. The cortex will then be affected and finally the medulla. Multiple tubercies will form (Fig. 15.3), coalesce together and may caseate and break down to give a cold abscess.

This may burst through the capsule of the gland into the tissues around it. Ultimately it may rupture through the skin producing a tuberculous sinus or ulcer.

Complications

1. Caseation and cold abscess formation which may burrow through the deep fascia or an overlying muscle so that it becomes bilocular and then called collar stud abscess (Fig. 15.4). Secondary infection of the abscess may occur and this makes the treatment more difficult.
2. A cold abscess is actually neither cold nor an abscess". It is not cold because clinically it is warm, but is in fact

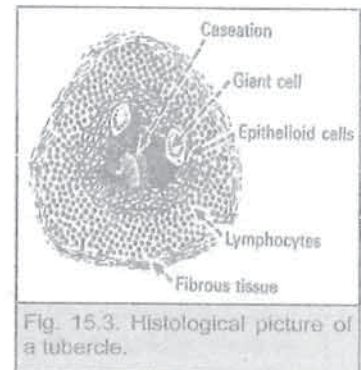


Fig. 15.3. Histological picture of a tubercle.

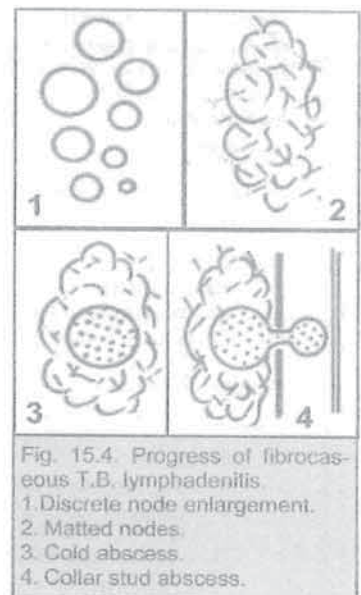


Fig. 15.4. Progress of fibrocasseous T.B. lymphadenitis.
1. Discrete node enlargement.
2. Matted nodes.
3. Cold abscess.
4. Collar stud abscess.

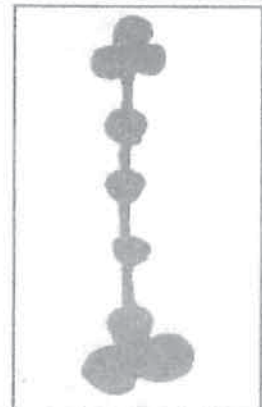


Fig. 15.5. Beaded cord between enlarged nodes.

colder than a pyogenic abscess. It is not an abscess because the contents are not pus but caseating material.

3. Sinus formation with a thin, cyanotic or bluish margin, undermined edges and a thin serous discharge. Secondary infection may be admitted along this sinus.
4. If the disease is left untreated, spread to the other groups of lymph nodes may occur.

Clinical picture

- Affected nodes are enlarged, usually not tender nor warm. They are firm or elastic in consistency and matted together. The consistency may be fluctuant due to breaking down. Attachment to the overlying skin may occur later when breaking down or a sinus is going to form. Enlargement of other groups of nodes must be sought for.
- An important diagnostic sign is the feeling of beaded cords between the different groups of nodes (Fig. 15.5). These cords represent thickened tuberculous lymphatics with intervening small tuberculous nodes along their course.
- A cold abscess, if present, is a soft, fluctuant swelling connected to the underlying caseating nodes which are felt by deep palpation. The overlying skin is at first normal, then it becomes dusky (not fiery red and not oedematous as in an ordinary pyogenic abscess). A cold abscess is slightly warm and slightly tender. The skin ultimately thins out before rupture, which ends in a sinus.

Investigations

1. Chest X-ray.
2. Tuberculin test serves as a good negative indicator.
3. Biopsy from the nodes will establish the diagnosis.
4. Aspiration of a cold abscess and examination of the pus microscopically, and guinea pig inoculation.
5. Smears from a sinus and examination for tuberculous organisms.

Treatment

1. Tuberculous lymphadenitis before caseation.

- a. The general condition of the patient is improved by good diet and vitamins.
- b. At least two antituberculous drugs are prescribed for at least 9 months. A combination of rifampicin and isonicotinic acid hydrazide is very efficient.
- c. Surgical excision is indicated for a single group of lymph nodes showing no response to medical treatment after a period of 6 months. A tuberculous abscess or sinus may be included with the nodes during excision.

2. Cold abscess

- a. Antituberculous drugs.
- b. Aspiration and injection of streptomycin solution. The rules of aspiration of a cold abscess should be followed to avoid sinus formation
 - i. The needle is inserted in a healthy part of the skin away from the abscess.
 - ii. The site of puncture should be in a non dependent part.
 - iii. The needle should also pass in a valvular manner. The points of entry through the skin and the abscess cavity should not be close and opposite to each other. To accomplish this, the needle pricks the skin, is advanced for some distance through the subcutaneous tissue, and is finally thrust deeply to enter the cold abscess cavity. Aspiration usually needs repetition every few days until the abscess dries up.
- c. Incision is indicated in the following conditions

- i. Secondary infection transforming the cold abscess into an acute pyogenic abscess.
- ii. If the abscess is imminent to rupture, it is incised, curetted, powdered with streptomycin and closed.
- d. Excision together with underlying lymph nodes is done if the nodes need excision.

3. Treatment of a tuberculous sinus

- a. General antituberculous treatment,
- b. Dressing with streptomycin powder every three days, until it closes.
- c. Excision with the underlying nodes, if resistant to conservative measures.

Blood-borne type

This type is more common in elderly people. The organisms reach lymph nodes via blood stream and so affect many groups of nodes in the body. The organisms enter the nodes through the arterial supply in the hilum and so the main affection is central in the medulla and peradenitis does not occur, and thus, there is no matUng.

As the patient is usually an adult of fair resistance, there is no breaking down, caseation nor cold abscess formation. The nodes do not reach a large size.

Clinical picture

The patient usually presents by multiple groups of enlarged lymph nodes which are not tender, not matted together, rubbery in consistency, and are always discrete (may be mistaken for Hodgkin's disease; hence the name lymphadenoid type of tuberculosis). No cold abscess or sinus is ever seen clinically in this type.

Treatment

After confirmation of diagnosis by biopsy, antituberculous treatment is prescribed.

Chronic lymphatic obstruction (lymphoedema)

Lymphoedema is a hypertrophic condition of the skin and subcutaneous tissues that is caused by chronic lymphatic obstruction. It affects the extremities, scrotum, external genitalia, and rarely the breasts. Lymphoedema of the lower limbs is the most common type. Another common example is post-mastectomy lymphoedema of the upper limb.

Aetiology

I. Secondary lymphoedema

1. Post-traumatic following

- a. Injuries as circumferential scars of the limbs.
- b. Operations as block dissection of regional lymph nodes.
- c. Burns at the site of lymph nodes.
- d. Irradiation of the regional lymph nodes.



Fig. 15.6. Filarial lymphoedema.

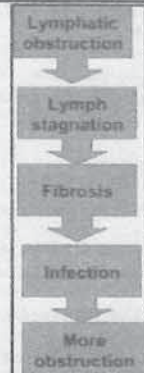


Fig. 15.7. Infection worsens oedema.



Fig. 15.8. Severe Filarial lymphoedema (elephantiasis).

2. Post-inflammatory

- a. Non-specific infection
 - i. Recurrent non-specific lymphangitis.
 - ii. Recurrent cellulitis due to evident focus of infection, e.g. interdigital infection and chronic leg ulcer.
 - iii. Post-erysipelas lymphoedema.
- b. Specific infections
 - i. Filarial (Fig. 15.6).
 - ii. Tuberculous (elephantiasis tuberculosa).
- c. Neoplastic
 - i. Primary affection of lymph nodes, as lymphomas.
 - ii. Secondary affection of lymph nodes by metastases.

II. **Primary lymphoedema** This is due to congenital malformations of the lymphatic vessels. Their types are shown in table 15.1

Table 15.1. Types of primary lymphoedema

	Lymphoedema congenita	Lymphoedema praecox	Lymphoedema tarda
Incidence	10%	80%	10%
Age	At or within 1 year of birth	Usually at adolescence	After 35 years
Sex	M > F	F > M	M = F
Site	Commonly bilateral and involves the whole leg	Commonly unilateral and below the knee	Usually unilateral

Pathology

Whatever the aetiology of lymphoedema, the pathological sequelae and the end result will be similar. Patients with lymphatic obstruction will suffer from two major problems

1. Stagnation of lymph leads to accumulation of large amounts of protein-rich fluids in the tissues. Proteins are irritant and, therefore, initiate foreign body reaction leading to fibrosis in the subcutaneous tissues and consequently more obstruction.
2. Recurrent attacks of lymphangitis lead to more lymphatic obstruction. There is a cascade of events which once starts proceeds in a vicious circle as shown in Fig. 15.7.

Pathological changes

1. Swelling: At first this is due to the accumulation of fluids, later the swelling is mainly due to fibrous tissue replacing the subcutaneous fat.
2. Early in the disease oedema is pitting. In later stages fibrosis hardens the skin and makes it non-pitting.
3. The skin changes: In primary lymphoedema skin changes are negligible. In longstanding secondary lymphoedema, there is thickening and hyperkeratosis of the skin. Lymphatic vesicles may appear in the skin. It is to be noted that although the skin is very much thickened, it is not liable to ulceration as the diffusion of nutrients is normal (compare with venous ulcers in post-phlebotic limbs).
4. In severe cases the skin develops huge bulges (Fig.15.8) and the condition is called 'elephantiasis'.

Filarial lymphoedema of the lower limbs

Aetiology and incidence

The most common cause of secondary lymphoedema of the lower limbs in Egypt is filarial infection with *Wuchereria bancrofti*. The disease is endemic in Egypt in areas near the Mediterranean, e.g. in Damietta and in some areas representing important stations at the desert edge, eg. Giza, Sharkiah and Assiut. The distribution of the lesion in the body is also variable and depends to some extent on the geographical locality, and the type of infecting filaria. One or both of the lower extremities with or without the scrotum are most commonly involved.

Pathology

1. The disease is due to obstruction of the lymph nodes by the adult worms leading to stagnation of lymph and dilatation of the lymph vessels.
2. Lymph stagnation leads to the accumulation of proteins in the subcutaneous tissues. These proteins will lead to cellular reaction in the form of plasma cells, eosinophils, lymphocytes, monocytes and polymorphnuclear leukocytes. Later on, fibrosis appears and collagen fibres are deposited with very little elastic tissue formation.
3. Recurrent attacks of lymphangitis occur leading to more fibrosis of the lymphatics.
4. Allergy to filarial antigens may have a role in the pathogenesis.

Differential diagnosis from other causes of chronic diffuse limb swelling.

1. Post-phlebitic limb.
2. Elephantiasis neurofibromatosis.
3. Congenital arterlo-venous fistula (local gigantism).
4. Other causes of lymphoedenia.

Diagnosis

Midnight blood film for microfilaria and positive intradermal skin test prove the diagnosis.

Treatment

1. Conservative treatment Mild and moderate cases of lymphoedema of the lower extremities are best treated with
 - a. Limb hygiene.
 - b. Elevation, massage and exercise.
 - c. Elastic stockings.
 - d. Diuretics, which are of controversial value.
 - e. Long acting penicillin 1,200,000 units every 3 weeks can be of value in stopping the recurrent attacks of lymphangitis.
 - f. Active filarial infection is treated by diethyl carbamazine (Chapter 7).
2. Surgical treatment. The operations are divided into two classes; none of them is completely reliable
 - a. The physiological operations are those designed to remedy the fault in the lymph drainage of the limb or to improve it in some way.
 - b. The excisional operations are designed to reduce the bulk of the limb by eradication of the swollen diseased tissues.

Physiological Operations

1. Enteromesenteric bridge operation. A segment of the ileum with its mesentery are separated and brought under the inguinal ligament. The mucosa of the bowel is removed and the opened bowel is sutured over the cut lymph nodes (Fig. 15.9A).

2. Omental flap operation A segment of the greater omentum is mobilized with intact blood supply and is transferred into the lower limb in an attempt to increase the lymphatic drainage (Fig. 15.9B).
3. Microlymphatic-venous anastomosis: The dilated obstructed lymphatic trunks are anastomosed to nearby veins.
4. Micro-lymphatic transfer operation Healthy lymphatic trunks are harvested from the normal limb and anastomosed to bypass the obstructed lymphatics. It is of special value in the treatment of post-mastectomy lymphedema of the upper limb.
5. Lympho-venous anastomosis: A lymph node is bisected and its distal surface is anastomosed to a nearby vein. It has the theoretical advantage that all afferent lymphatics to the node may be included in the shunt.

Excisional operations

1. Charles' operation entails excision of the skin and subcutaneous tissues and covering the limb by split skin graft (Fig. 15.10A).
2. Sistrunk's operation includes excision of an ellipse of skin and subcutaneous tissues and closure of the defect. The aim is to decrease the bulk of the limb at one side. This can be repeated on the other side at a later date (Fig. 15.10B).

Physiological and excisional operations

Thompson's (Swiss roll) operation consists essentially of excision of subcutaneous tissue and then implantation of a shaved flap of skin between the muscles near the deep lymphatic vessels so that physiological drainage may be improved (Fig. 15.11). This operation is not commonly performed nowadays.

Neoplastic disorders

1. Lymphomas. These form a group of malignant neoplasms that arise in lymph nodes or extranodal lymphoid tissue.
 - a. Hodgkin's disease (HD)
 - b. Non-Hodgkin's lymphoma (NHL)
 - c. Burkitt's lymphoma
2. Leukaemias
3. Secondary deposits

Hodgkin's disease (HD)

Hodgkin's disease is the most frequently occurring lymphomas.

Pathology

Macroscopically the disease affects lymph nodes and extranodal lymphoid tissue in the spleen, liver, and bone marrow. It usually starts in cervical lymph nodes. The affected nodes are enlarged, discrete, rubbery, and have a pink colour. Usually there is a large lymph node in the centre with smaller lymph nodes around it.

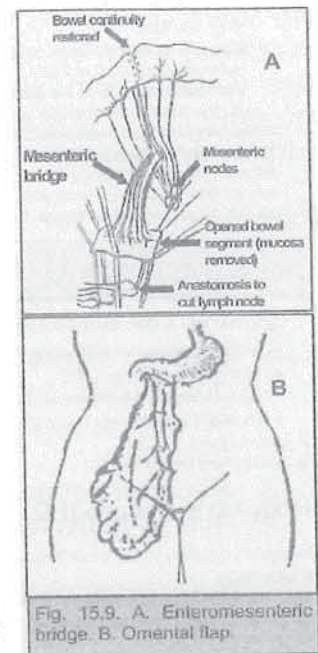


Fig. 15.9. A. Enteromesenteric bridge. B. Omental flap.

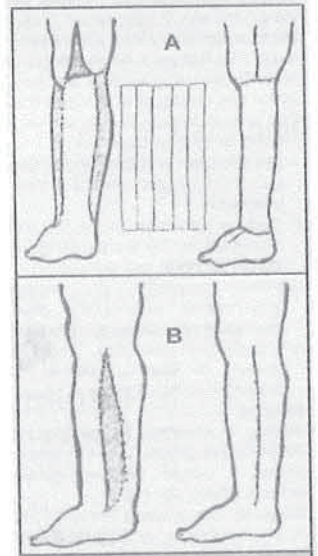


Fig. 15.10. A. Charles operation. B. Sistrunk's operation.

Microscopically there are variable densities of lymphocytes, yet the diagnostic feature is finding the characteristic Dorothy-Reed Sternberg cells. These are giant cells that have an even number of nuclei (2 or 4) which are arranged in a mirror image manner (Fig 15.12). There are four known histological types of Hodgkin's disease. In descending order of prognosis these are

1. Lymphocyte predominance. Giant cells are rare, but there are abundant lymphocytes and histiocytes. This type carries the best prognosis.
2. Nodular sclerosis. This is the commonest histological variety and is characterized by the presence of broad fibrous bands that disrupt the lymph node architecture.
3. Mixed cellularity. A heterogenous cellular component of lymphocytes, eosinophils, plasma cells, and the giant Reed Sternberg cells characterizes this type of Hodgkin's disease.
4. Lymphocyte depletion. There is considerable fibrosis, variable amount of Reed Sternberg cells, and few lymphocytes. The prognosis is the worst of all types.

Staging

Staging is essential for planning treatment and for having an idea about the prognosis. The Ann Arbor staging system is in common use.

- Stage I Single involved lymph node group (I), or a single extralymphatic site (IE).
- Stage II Two or more involved lymph node groups limited to one side of the diaphragm or a solitary extralymphatic site and one or more lymph node areas on the same side of the diaphragm (IIE).
- Stage III Involvement on both sides of the diaphragm with or without splenic involvement.
- Stage IV Extralymphatic spread including liver, lung, bone marrow, skin, gut, and central nervous system involvement.

All stages are subdivided into either

- (A) No systemic symptoms.
- (B) One or more of the three systemic symptoms, fever, night sweats, and weight loss of more than 10% in six months.

Clinical features

- Caucasians are affected by Hodgkin's disease more than the other races.
- No age is exempt, yet the disease shows two age peaks, the first is between 15 to 35 years, and the other is above 50 years.
- The usual presentation is by painless progressive enlargement of the cervical lymph nodes (Fig. 15.13). With progress of the disease other node groups in the neck, axillae, groin, mediastinum, and abdomen are affected. These nodes are enlarged, discrete, non-tender, and rubbery in consistency.
- Splenomegaly and hepatomegaly may also be present.

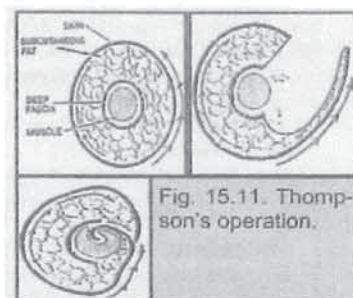


Fig. 15.11. Thompson's operation.

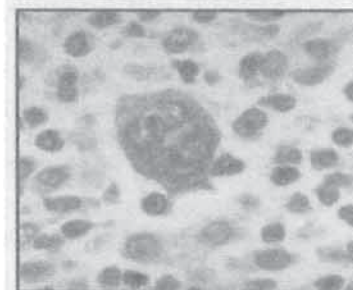


Fig. 15.12. Reed Sternberg cell.



Fig. 15.13. The commonest site for Hodgkin's disease is the cervical lymph nodes.



Fig. 15.14. The commonest site for Burkitt's lymphoma is the jaw of an African child.

- Some patients exhibit systemic manifestations in the form of fever, night sweats, weight loss, pruritus, anaemia, and jaundice. Sometimes a characteristic intermittent fever which lasts for a few days followed by a remission for few weeks occurs (Pel-Ebstein fever).
- Immediate pain may occur in diseased areas after drinking alcoholic beverages.

Investigations

The aim of investigations is a dual one, viz, to diagnose the cause of lymphadenopathy, and to detect the extent of the disease (staging).

1. **Full blood picture** usually shows no abnormality, but the following are sometimes present
 - a. Anaemia.
 - b. Oesinophilia or lymphopenia.
 - c. High ESR.
2. **Alkaline phosphatase** is elevated in cases with bone or liver involvement. Other liver function tests may be affected with advanced liver disease.
3. **Lymph node biopsy** is the cornerstone of diagnosis. The neck is the preferred site of biopsy. Inguinal lymph nodes are better avoided as they are commonly enlarged as a result of chronic nonspecific lymphadenitis. The use of general anaesthesia is preferred to allow making a liberal incision to take a whole lymph node without the rough manipulations that may damage its architecture.
4. **Chest X-ray and CT scan** allow detection of intrathoracic disease.
5. **Abdominal ultrasound and CT scan** can detect enlargement of the liver, spleen and abdominal lymph nodes.
6. **Staging laparotomy**. When a patient with Hodgkin's disease is diagnosed clinically and by the above mentioned investigations to have an early stage of Hodgkin's disease, the treating doctor should make sure that there is no intra-abdominal disease, as this will have implication on the strategy of treatment. Indications for staging laparotomy, therefore, include stages IA, IB, and IIk Patients with advanced disease are not candidates for the operation as they will certainly receive chemotherapy.

The operation includes

- (a) Splenectomy.
- (b) Biopsy of both liver lobes.
- (c) Biopsy of all intra-abdominal lymph node groups, which are marked by metal clips to help future localization by the radiotherapist.
- (d) Bone marrow biopsy from the iliac crest.
- (e) In females the ovaries and Fallopian tubes are fixed in the middle line behind the uterus to guard them against irradiation when treating the iliac nodes.

In addition to the benefit of staging, removal of the spleen obviates the need for its irradiation and, hence, spares the left kidney and lung the hazards of radiation injury. Lately, however, the operation of staging laparotomy is dropping out of favour in many centres because of the following two facts:

- (a) The high accuracy of CT scan, and the increasing availability of MRI,
- (b) The risk of overwhelming post-splenectomy infection (OPSI), that is particularly common and fatal in children.

Treatment

A multidisciplinary approach that includes the oncologist, radiotherapist, and surgeon, gives the best result. As a general rule, early localized disease is controlled

mainly by radiotherapy, while widespread systemic disease is mainly treated by chemotherapy. The role of the surgeon is limited to lymph node biopsy and to staging laparotomy, whenever indicated.

The following is a simplified scheme of treatment.

- Stage IA. IB and IIA are treated by radiotherapy applied to the affected group of lymph node, as well as to adjacent groups.
- Stage IIB is treated by radiotherapy and 6 cycles of combination chemotherapy.
- Stage III and IV are treated by 12 cycles of chemotherapy. Radiotherapy is used as a supplement to control bulky cervical nodes.

The MOPP chemotherapy regimen is the one in common use and is formed of mustine (nitrogen Mustard), Oncovin (vincristine), Procarbazine, and Prednisone.

Prognosis

Prognosis depends upon the histological type, and the stage. On average an 80% 5 years survival is obtained with proper treatment. For stage IA with lymphocytic predominance the result approaches 100%. Stage IIIA has 80% 5 years survival.

Non-Hodgkin's Lymphoma (NHL)

Non-Hodgkin's lymphoma comprises a heterogenous group of neoplasms that arise from a monoclonal proliferation of a malignant cell of lymphoid origin.

Aetiology

The exact aetiology is not known, but the disease has a higher incidence with the following conditions

1. Sjogren's disease, and benign lymphoepithelial lesions of the salivary glands.
2. Immune deficiency as with AIDS and with prolonged immunosuppression after organ transplantation.
3. May be related to infection with human T-cell leukemia-lymphoma virus (HTLV-I).
4. Systemic lupus erythematosus.

Classification

Classification is based on the cell of origin

1. B cell lymphoma (80-85%) is further classified into
 - a. Small cell lymphoma.
 - b. Large cell lymphoma.
 - c. Mixed small and large cell lymphoma.
 - d. Immunoblastic lymphoma.
2. T cell lymphoma.
3. Lymphoblastic lymphoma.
4. Histiocytic lymphoma.

In each type the cells may be arranged in a nodular or a diffuse pattern.

Clinical features

1. Lymph node enlargement is similar to that of Hodgkin's disease. Progression of lymph node enlargement, however, does not follow an orderly anatomical pattern as in Hodgkin's disease.
2. NHL is more likely to present in extranodal sites than Hodgkin's lymphoma.
3. NHL patients come under the surgeon's care when the disease affects the gut.
 - a. Gastric lymphoma produces manifestations that are similar to carcinoma.

- b. Intestinal lymphomas may produce intestinal obstruction, bleeding, or perforation.
 - c. Staging laparotomy is rarely indicated.
 4. Mycosis fungoides is a variant of NHL in which skin eruption is the first of the disease manifestations.
 5. The disease is likely to be disseminated at the time of presentation.
- Staging** of NHL follows the same criteria as those of Hodgkin's disease.

Treatment

- Radiotherapy and combination chemotherapy constitute the main lines of treatment according to the stage of the disease. The commonly used drugs are cyclophosphamide, adriamycin, vincristine, prednisone, and bleomycin.
- Surgery is needed to deal with gastric and intestinal disease by gastrectomy and by intestinal resection. Surgery is followed by radio and chemotherapy. The prognosis of gastric lymphoma is better than that of gastric adenocarcinoma.

Burkitt's lymphoma

This form of lymphoma is common among children living in the eastern part of Africa which is known to be endemic for malaria.

Aetiology

The exact aetiology is not known yet Burkitt's lymphoma is thought to be related to infection with the Epstein Barr (EB) virus. Malaria may have a role in paving the way for the EB virus to induce the disease.

Pathology

Burkitt's lymphoma is a malignant tumour of the B lymphocytes. In most cases it affects the jaw.

Clinical features

The disease affects children below the age of 12 years, with a male predominance. The usual presentation is that of a child who has a painless progressively enlarging jaw swelling (Fig. 15.14). This swelling distorts the face, may displace the eye, and partially occludes the mouth. The lymphoma also affects the kidneys, retroperitoneal tissues, ovaries, long bones, and central nervous system.

Treatment

Treatment is by chemotherapy using a combination of cyclophosphamide and cytosine arabinoside. There is usually good initial response, which should be followed by further chemotherapy cycles to prevent recurrence.

Leukaemias

Unlike lymphomas, leukaemias are characterized by generalized enlargement of the lymph nodes, splenic and liver enlargement together with a marked increase in the total leucocytic count mostly made of immature forms. Acute leukaemia is of no interest to the surgeon, but the chronic forms are important in the differential diagnosis of lymphadenopathy, splenomegaly and gastrointestinal haemorrhage.

Chronic myeloid leukaemia affects both sexes equally between the ages of 35 and 70 years. The onset is insidious with anaemia, loss of weight, spontaneous haemorrhages, marked splenomegaly, hepatomegaly and generalized lymphadenopathy

but the nodes are slightly enlarged and discrete. Blood examination shows a progressive anaemia with a great increase in the white count up to 1000,000/ul, 80-90% of the white cells being of the granular series. The disease is invariably fatal, and the average duration of life is 2-3 years.

Chronic lymphatic leukaemia affects elderly people, especially males. There is moderate enlargement of all lymph nodes and lymphoid tissues in the body, but the spleen is much smaller than in myeloid leukaemia. Blood examination shows a moderate anaemia with a great increase in the white count up to 500,000/ul, 80-90% being lymphocytes. Death usually occurs in 10 years. In elderly patients the disease tends to progress very slowly.

Secondary Deposits

Secondary carcinoma is very common in the lymph nodes due to lymphatic extension from a primary lesion in their drainage areas. Such deposits are especially common in the neck from epithelioma of the mouth, pharynx, nose and scalp, but sometimes the primary growth lies in a hidden site and may be overlooked, as in the ear, hypopharynx, nasal sinuses, nasopharynx, bronchi, stomach and testis.

Clinically, the affected nodes are stony hard, painless and mobile at first, but soon they become fixed and painful and may ulcerate and fungate through the skin.

Treatment

- In early cases, removal of the primary tumour with radical block dissection of the affected nodes is the ideal treatment.
- Late cases with inoperable primary or fixed nodes are treated by palliative radiotherapy or chemotherapy.

MUSCLES, TENDONS AND FASCIÆ

Carpal tunnel syndrome

This is the commonest type of nerve compression syndromes.

Anatomical background

The median nerve is compressed as it passes through the carpal tunnel, in front of the wrist. The tunnel is bounded posteriorly by the carpal bones, and anteriorly by the flexor retinaculum. In addition to the median nerve, the carpal tunnel houses the eight tendons of the flexor digitorum superficialis and profundus, enclosed in the ulnar bursa, and the flexor pollicis longus tendon, enclosed in the radial bursa (Fig. 16.1 and 17.1),

After passing deep to the retinaculum, into the hand, the median nerve supplies the following: (Fig. 16.2)

▪ Muscles

- Three muscles of thenar eminence.
- Lateral two lumbricals.

▪ Skin

- Lateral three and half fingers.

The palmar branch of median nerve arises above the wrist and passes superficial to the flexor retinaculum.

Aetiology

- This disorder affects essentially middle aged females.
- It may be precipitated by
 - Pregnancy.
 - Moedema.
 - Acromegaly.
 - Rheumatoid arthritis involving the synovial sheaths of the flexor tendons leading to increased tension under the retinaculum.
- May be idiopathic.

Clinical features and investigations

- The early symptom is pain in the thumb and lateral three fingers that gets worse by night.
 - The little finger is never affected in this syndrome.
 - The palm is not affected as the palmar branch of median nerve escapes compression.
 - Pain is increased by fully flexing the wrist.
 - Relief of pain is obtained by injecting the tunnel with lidocaine and corticosteroids, and by splinting the wrist at night.
- Late manifestations
 - Paraesthesia and altered sensibility of the skin supplied by the median nerve.
 - Weakness and atrophy of the thenar eminence muscles.

CHAPTER CONTENTS

- Carpal tunnel syndrome
- Dupuytren's contracture
- Volkmann's Ischaemic contracture
- Chronic tendinitis Chronic bursitis



Fig. 16.1. Median nerve passes deep to flexor retinaculum together with the long flexor tendons.

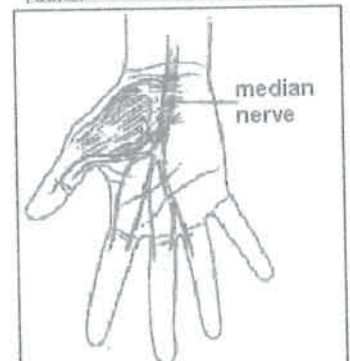


Fig. 16.2. Distribution of median nerve after entering the hand.

Dupuytren's contracture

- The condition is commonly confused with the hand pain produced by cervical spondylosis.
- In the case of carpal tunnel syndrome a median nerve conduction test reveals a delay at the carpal tunnel.

Treatment

Surgical division of the flexor retinaculum (Fig. 16.3) produces gratifying results. After decompression, the median nerve should be inspected, and if it still shows a constriction, release of its tight epineurium is required.

Dupuytren's contracture

This is an idiopathic disorder that is also known as "palmar fascitis".

Pathology

The disease is characterized by progressive thickening and contraction of the palmar aponeurosis.

Aetiology

- Idiopathic
- The disease is known to have a higher incidence in cirrhotics, alcoholics, epileptics under phenytoin treatment, and diabetics
- Some cases are familial.
- Males are affected ten times more than females.

Clinical features

- Dupuytren's contracture affects primarily the medial side of the palmar aponeurosis.
- The disease is bilateral in about half the cases.
- It starts as a nodule at the base of the ring or little finger (Fig. 16.4). This is followed by contracture which throws the finger or fingers into flexion.
- The skin gets adherent to the fascia and contracts as well.
- The other fingers progressively become involved.
- In late cases the capsules of the metacarpo-phalangeal and proximal interphalangeal joints also become involved.
- The flexion deformity affects the metacarpophalangeal and the proximal interphalangeal joints. The flexion deformity is not lessened by flexing the wrist joint.
- The distal interphalangeal joints are free.
- Palpation of the palm reveals a firm, irregular shaped nodule, 1-2 cm proximal to the base of the ring finger. Tight strands can be felt running from the nodule to the bases of the ring and little fingers and proximally to the flexor retinaculum.

Treatment

- Early cases can be controlled by physiotherapy.



Fig. 16.3. Division of flexor retinaculum releases the compressed median nerve.



Fig. 16.4. Dupuytren's contracture.



Fig. 16.5. Supracondylar fracture may injure brachial artery

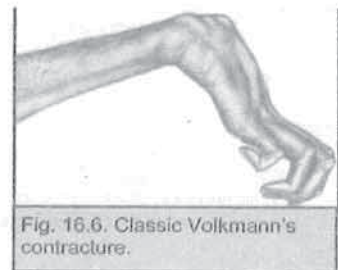


Fig. 16.6. Classic Volkmann's contracture.

- Surgery is indicated for late cases. The scope of surgery varies from minor subcutaneous fasciotomy to an extensive operation that includes excision of the aponeurosis, joint capsulotomy and skin grafting to cover the resulting defect after stretching the fingers.

Volkmann's ischaemic contracture

This is a condition of fibrosis and shortening of the muscles of the front of the forearm as a result of acute ischaemia and infarction.

Aetiology

- Acute ischaemia of the muscles of the front of the forearm is usually a sequel of brachial artery compromise after injuries around the elbow region.
 - Supracondylar fracture in children is a well known cause (Fig. 16.5). The ischaemia is more commonly caused by a tight plaster cast applied to treat the fracture rather than due to the fracture itself.
 - Other less frequent causes include direct brachial artery injury, and arterial embolism.
- Acute muscle ischaemia results in infarction after 6-12 hours. Muscle fibrosis and shortening develop within a few months.

Clinical features

- Early, while the arm is immobilized for treatment of the fracture
 - The patient complains of pain in the forearm muscles and hand.
 - Passive extension of the fingers is painful and limited.
 - The fingers and hand are pale, cold and the capillary circulation may be sluggish.
 - The radial pulse may be absent.
 - If the median nerve is ischaemic, there may be paraesthesia or severe burning pain in its distribution.
 - The appearance of these features in a patient with a fracture or after application of a plaster cast is very serious, because it is at this stage that ischaemia can be corrected and the muscles saved.
- Established contracture
 - Pain is lessened but the patient will present by the typical deformity (Fig 16.6).
 - The forearm and hand appear wasted.
 - All the finger joints are flexed (claw hand).
 - Extension of the fingers is limited but improves as the wrist is flexed (compare with Dupuytren's contracture). Hand grip is weak. An important diagnostic feature is that all the muscles have some function, whereas when a claw hand is caused by a nerve lesion, some of the muscles will be completely paralysed.

Differential diagnosis of claw hand

1. Volkmann's ischaemic contracture.
2. Combined median and ulnar nerve injuries.
3. Lesions affecting the lower roots or medial cord of the brachial plexus, e.g. Klumpke's paralysis, and malignant infiltration of the brachial plexus.
4. Advanced rheumatoid arthritis.

5. Spinal cord lesions as syringomyelia and poliomyelitis.

Prevention

Prompt restoration of muscle blood supply before the development of infarction is the key to prevention.

- Reduction of fractures and dislocations, release of tight plaster cast and removal of constrictions can restore arterial pulsation.
- If the radial pulse does not return, the brachial artery is explored, and the injured segment is excised and the artery is reconstructed by a vein graft.

Treatment

1. Early cases. Physiotherapy by gradual stretching and splinting can maintain the muscle length.
2. Late cases. Muscle slide operation is done. The common flexor origin is detached from the medial epicondyle and is fixed at a lower level.

Chronic tendonitis

Rotator cuff (supraspinatus) tendonitis

- This is the commonest cause of shoulder pain with limitation of movement. Any of the rotator cuff muscles may be involved, yet the commonest is the supraspinatus tendon (Fig. 16.7).
- The usual cause is repeated trauma from sports or occupational activities. Inflammation often extends to affect the subacromial bursa.
- Active abduction is particularly painful when the shoulder moves between 60 and 120 degrees. This is because the inflamed rotator cuff and bursa are compressed beneath the acromion. For this characteristic feature the condition is also known as "painful arc syndrome".
- Treatment is conservative using nonsteroidal anti-inflammatory agents. Shoulder immobilization, for a few days only, relieves the pain of acute exacerbation. This should be followed by gradual active exercise to restore the full range of shoulder movement. Resistant cases can benefit from local injection of corticosteroid lidocaine mixture.

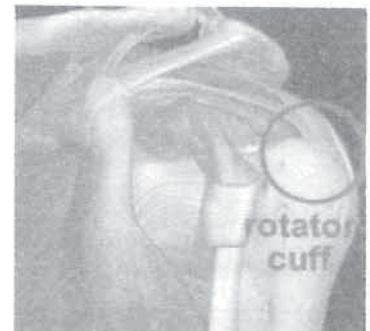


Fig. 16.7. Supraspinatus tendon is affected at its insertion.

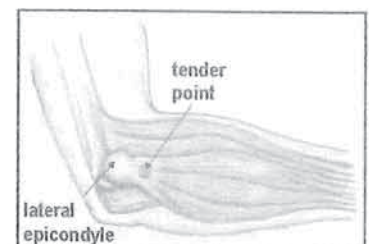


Fig. 16.8. Tennis elbow.

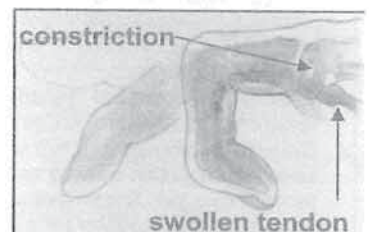


Fig. 16.9. Trigger finger.



Fig. 16.10. DeQuervain's synovitis.

Tennis elbow

- The patient complains of pain in the elbow at rest and in particular when he uses his hand. The attachment of extensor muscles of the forearm to the lateral epicondyle is tender (Fig. 16.8). The condition is due to direct trauma or repeated athletic activity.
- Treatment is by rest. In resistant cases local injection of corticosteroid and local anaesthetics is usually successful.

Stenosing tenovaginitis

Stenosing tenovaginitis is caused by a fibrous stricture in a tendon sheath. There are two common examples, and both affect the hand.

Trigger finger

- This condition affects usually middle aged women and young children.
- The fibrous flexor sheath at the level of the metacarpophalangeal joint is thickened producing pain on tendon movement, and local tenderness (Fig. 16.9).
- When the finger is flexed the part of the tendon proximal to the constriction may get swollen. This bulge interferes with extension of the finger, locking it in flexion. Attempts to extend the finger may require passive assistance which will cause the tendon to snap through the strictured area. This is likened to the snap of the trigger of a pistol, hence the name "trigger finger".
- The thickening of the tendon or tendon sheath can be felt at the level of the head of the metacarpal bone.
- The condition is treated by division of the constricting fibrous flexor sheath.

DeQuervain's synovitis

- At the point where the abductor pollicis longus and extensor pollicis brevis tendons cross the wrist, their sheath is inflamed (Fig. 16.10) producing pain and limitation of movement.
- Active and passive movements of the thumb exaggerate the pain. The condition is treated by division of the constricting tendon sheath.

Chronic bursitis

Bursae are fluid-filled cavities, lined with flattened epithelium similar to synovium. There are two types of bursae, the anatomical, and the adventitial.

Inflammation of anatomical bursae

Anatomical bursae are normally found to allow easy movement between tendons, bones, and skin.

Chronic inflammation of such bursae usually results from repeated friction, and presents with mildly painful cystic swellings. Crepitus is a grating sensation that may be noticed by the patient if the bursa lining is rough, or if the fluid contains small loose fibrinous particles.

Common site of these bursitis include

1. Prepatellar bursitis (House-maid's knee). The bursa is situated over the lower half of the patella behind the skin.
2. Infrapatellar bursitis (Clergyman's knee). The bursa lies between the ligamentum patella and the head of the tibia and when distended, it gives a fluctuating swelling on either side of the ligamentum.
3. Olecranon bursitis (student's elbow). The bursa lies between the skin and the olecranon (Fig. 16.11).

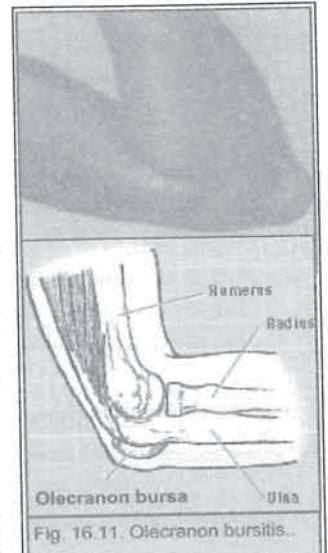


Fig. 16.11. Olecranon bursitis.

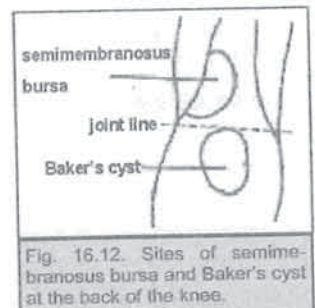


Fig. 16.12. Sites of semimembranosus bursa and Baker's cyst at the back of the knee.

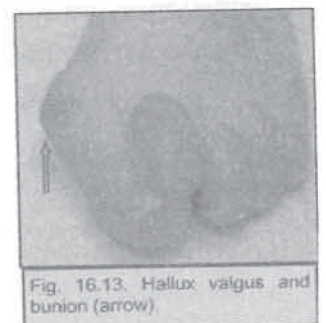


Fig. 16.13. Hallux valgus and bunion (arrow)

4. **Semimembranosus bursa.** This bursa exists between the semimembranosus tendon and the postero medial aspect of the femoral condyle. The patient usually a young or middle-aged adult complains of a swelling behind the knee joint, that may interfere with knee movements. Examination reveals a cystic swelling on the medial side of the popliteal fossa just above the joint level. Contraction of the semimembranosus muscle during flexion of the knee makes the swelling diminish in size. Semimembranosus bursa may be confused with a condition called "Baker's cyst" which is a pulsion diverticulum of the knee joint affecting patients with rheumatoid arthritis or osteoarthritis. It occurs more in elderly patients and presents as a cystic swelling below the level of the knee joint and deep to the gastrocnemius muscle (Fig. 16.12). The characteristic finding of this cyst is that it can reduce into the joint. This is elicited clinically by compression of the cyst where a swelling appears at each side of the Patella.

Adventitious bursae

Adventitious bursae develop in any site of friction between two layers of tissue. A known example is the "bunion" which develops between the skin and the head of the first metatarsal, particularly with the hallux valgus deformity (Fig. 16.13).

Treatment

If friction can be abolished, bursitis may regress, otherwise the treatment is excision and correction of any underlying deformity (Chapter 55).

Soft tissue sarcomas

- Soft tissue sarcomas are malignant tumours arising in the extra skeletal connective tissue.
- They are relatively uncommon and account for around 1% of all malignant tumours.
- The majority arise in the extremities and pelvic girdle. They also arise in the trunk and in the retroperitoneal tissues.
- These tumours occur in all age groups including children, yet they are commonest in the fifth and sixth decades of life.

Aetiology

The exact aetiology is poorly understood, but some disorders are associated with certain types of soft tissue sarcomas.

- Following irradiation for other malignancies, e.g. Hodgkin's disease.
- Lymphangiosarcoma may develop in the chronic post mastectomy arm oedema.
- Neurofibrosarcoma may develop in patients with von Recklinghausen's disease.

Pathology

Types and histological picture

Most soft tissue sarcomas arise from primitive multipotential mesenchymal (connective tissue) cells, which differentiate during the process of neoplastic transformation to form one of the following types

1. Malignant fibrous histiocyoma.
2. Liposarcoma.
3. Rhabdomyosarcoma.
4. Synovial sarcoma.
5. Malignant nerve sheath tumour.
6. Leiomyosarcoma.
7. Fibrosarcoma.
8. Angiosarcoma.

Gross appearance

- A characteristic feature is the presence of a well defined false capsule from which the contents can be easily enucleated. The capsule, however, is an integral part of the tumour, infiltrated with malignant cells which spread well beyond it. Enucleation of the tumour from within the pseudocapsule is, therefore, an inadequate and obsolete treatment.
- The cutsection is commonly fleshy, and shows areas of necrosis and haemorrhage.

Grade of malignancy

- Soft tissue sarcomas express variable grades of malignancy that are assessed according to certain histological criteria which include extent of mitosis, extent of necrosis, cellular anaplasia and pleomorphism. The tumour is then judged to be low, intermediate, or high grade. Histological grading has more influence on prognosis than the type of the tumour.
- DNA ploidy is also used to grade these tumours. A normal cell is euploid, i.e. it contains 46 chromosomes (2n). Malignant cells, on the other hand, are usually polyploid (contain 4n, 8n, ... etc.), and aneuploid (bizarre chromosomal content). Using a "flow cytometer", the deviation of a tumour from the normal ploidy can be assessed, thus determining the tumour aggressiveness.

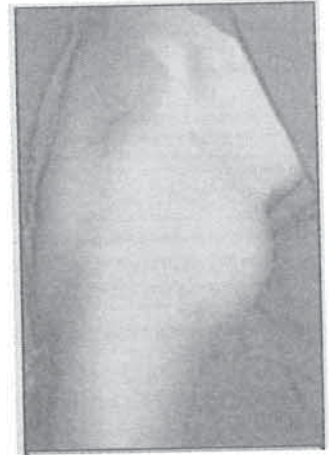


Fig. 16. 14. Soft tissue sarcoma of medial side of right thigh.

Spread

- Local spread occurs first within the musculofascial compartment in which it arises. Fascial septa resist spread temporarily, after which extracompartmental spread occurs.
- Blood-born spread from high grade tumours goes mainly to the lungs.
- Lymphatic spread is unusual.

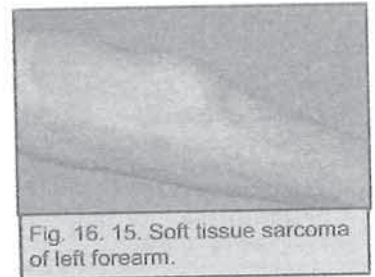


Fig. 16. 15. Soft tissue sarcoma of left forearm.

Clinical features

- The majority of patients complain of a swelling (Fig. 16.14 and 16.15), which has been gradually enlarging over several months.
- The swelling is often painless and without disability, so there may be a substantial delay before presentation, the tumour being remarkably large when first examined.
- The tumour consistency may be soft or firm, depending on the amount of deposited collagen.

Differential diagnosis

1. Benign soft tissue tumours, e.g., a lipoma.
2. Deep seated haematoma.
3. Malignant lymph nodes.
4. Bone tumours.
5. Tumour like conditions of the soft tissues.
 - Fibromatosis. The best known example is the desmoid tumour (Paget's recurrent desmoid tumour). It occurs most often in the rectus sheath and the external oblique aponeurosis of middle aged women. It grows at a very slow rate, and has a tendency for recurrence after an apparently adequate excision. That is

why it has been considered, in the past, to be a low grade soft tissue sarcoma. Wide surgical excision is the only treatment.

- Nodular and proliferative fasciitis.
- Proliferative myositis and myositis ossificans.

Investigations

Tissue diagnosis

It is essential to obtain histological confirmation of the diagnosis before planning treatment.

- Fine-needle biopsy is done under local anaesthesia. It is a simple and safe procedure that does not interfere with subsequent resection.
- Open biopsy is necessary when a firm diagnosis cannot be reached after repeated fine-needle biopsy.

Imaging

- CT scan or MRI of the tumour area is the investigation of choice, providing excellent definition of the extent of the tumour and its relationship to muscle groups (Fig. 16.16).
- Chest X-ray and CT scan to detect pulmonary metastases.

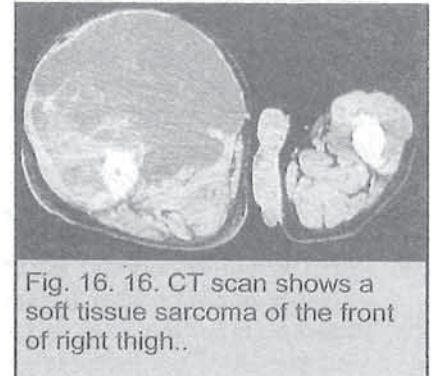


Fig. 16. 16. CT scan shows a soft tissue sarcoma of the front of right thigh..

Treatment

- (A) Operable cases: A combination of radical surgical excision and adjuvant (complementary) radiotherapy is the standard treatment. The extent of surgery depends on the extent of tumour invasion.
 - If the tumour is still confined to a muscle compartment of a limb, the whole compartment is removed within its fascial envelope. Enucleation of the tumour from within its pseudocapsule is followed by recurrence. Major reconstruction of skin and soft tissue defects is commonly required.
 - If radical resection is judged to leave a useless limb, amputation is indicated.
- (B) Inoperable cases: Patients with pulmonary metastases, and those with huge retroperitoneal sarcomas that are adherent to important irremovable structures, are considered to be beyond cure.
 - Combination chemotherapy, though not so effective, is the main line of palliation.
 - Palliative surgical excision may be added.

THE HAND AND FOOT

Surgical anatomy of the hand

The hand is a perfect piece of machinery that is adapted to perform highly skilled functions. These are

1. Touch.
2. Precision handling.
3. Power grip.
4. Pincer grip, e.g. holding a key making use of the opposition function of the thumb.
5. Expression e.g. writing, signalling, and hand shaking.

The hand is formed of a bones, joints, tendons, intrinsic small muscles, fascial septa that form compartments, nerves, vessels, and skin.

Bones

Carpus. The carpus is formed 8 small bones that arranged in two rows, the proximal of which articulates with the lower parts of the radius and ulna at the wrist joint.

Metacarpus. There are five metacarpal bones that articulate with the phalanges at the metacarpophalangeal joints.

Phalanges. The thumb is formed of two phalanges, while each of the other four fingers is formed of three.

Tendons

Tendons of long muscles cross the wrist ventrally (flexors) and dorsally (extensors) to move the wrist and fingers.

Flexor tendons. The long flexor tendons arise from two muscles, the flexor digitorum superficialis (sublimis) and the flexor digitorum profundus. These have a special arrangement at their insertion. Each superficialis tendon splits opposite the middle phalanx forming a tunnel, and gains insertion at the sides of the anterior surface of this phalanx. The profundus tendon passes through this tunnel and crosses the distal interphalangeal joint to be inserted in the front of base of the distal phalanx (Fig. 17.1). Therefore, the superficialis muscle does not flex the distal interphalangeal joint, while the profundus can move all joints which it crosses.

Synovial sheaths. To allow free frictionless movement of the hand, the flexor and extensor tendons are enclosed in two layers of synovium separated by fluid. Opposite each finger, the flexor tendons are enclosed in a digital synovial sheath, which in turn is covered by a fibrous

CHAPTER CONTENTS

- Surgical anatomy of the hand
- Hand infection
- Ingrowing toe nail
- Hand injuries

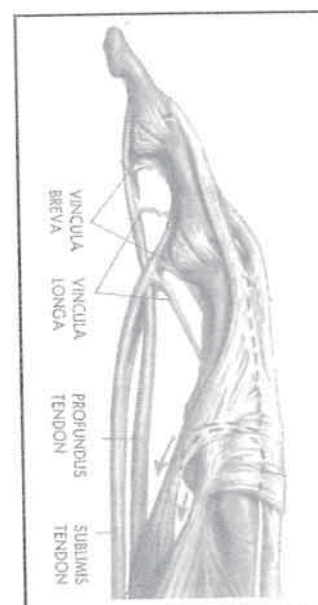


Fig. 17.1. Arrangement of long flexor tendons of fingers.

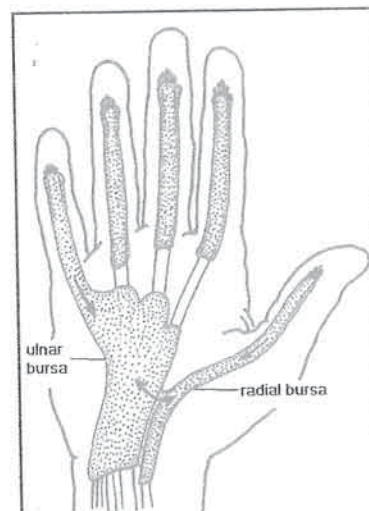


Fig. 17.2. Radial and ulnar bursae.

flexor sheath. The flexor pollicis longus has a separate synovial covering that is called the radial bursa, while in the palm and distal forearm, the superficial and deep flexor tendons of the other fingers are enclosed in the ulnar bursa. The radial and ulnar bursae commonly communicate, while the latter communicates with the digital synovial sheath of the little finger (Fig. 17.2). These facts are reflected on the spread of tendon sheath infections.

Intrinsic muscles

The thenar and hypothenar muscles serve flexion, abduction, and opposition of the thumb and little finger. The thumb has an additional adductor pollicis muscles.

The dorsal interossei abduct, while the palmar interossei adduct the metacarpo-phalangeal joints.

The lumbricals arise from the flexor digitorum profundus tendons and are inserted with the long extensor tendons, in the extensor expansion. The lumbricals and the interossei place the hand in the writing position, i.e. flexion of the metacarpo-phalangeal, and extension of the interphalangeal joints. The thumb has no lumbrical.

Flexor retinaculum

The flexor retinaculum extends transversely across the anterior concavity of the carpus, converting it into a tunnel (Fig. 17.3). The long flexor tendons with their synovial covering, and the median nerve pass through this carpal tunnel to reach the hand.

Fascial spaces

The mid-palmar space lies between the flexor tendons of the little, ring and middle fingers anteriorly and the fascia covering the interosseous muscles posteriorly. Medially a fibrous septum separates it from the hypothenar eminence.

Laterally a strong fibrous septum extends from the palmar fascia to the middle metacarpal bone and separates the mid palmar space from the thenar space (Fig. 17.4).

Nerves

As mentioned, the median nerve passes into the hand deep to the flexor retinaculum. It provides innervation to the thenar muscles (except the adductor pollicis), and the first lumbrical. The median nerve also provides sensory branches to the lateral two thirds of the palm skin and the anterior surface of the lateral three and half fingers. The ulnar nerve, on the other hand, is the main motor supply of the small muscles of the hand. It supplies the rest of the intrinsic muscles and provides sensation to the front and back of the medial third of the hand and the medial one and half fingers. The radial nerve has no motor branches in the hand, yet it supplies the rest of skin of the dorsum of the hand and fingers. Cutaneous innervation of the hand is shown in Fig. 17.5.

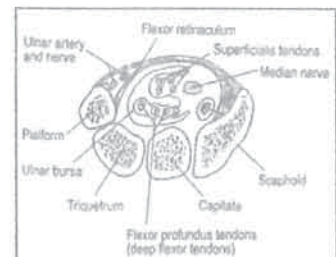


Fig. 17.3. Flexor retinaculum and carpal tunnel.

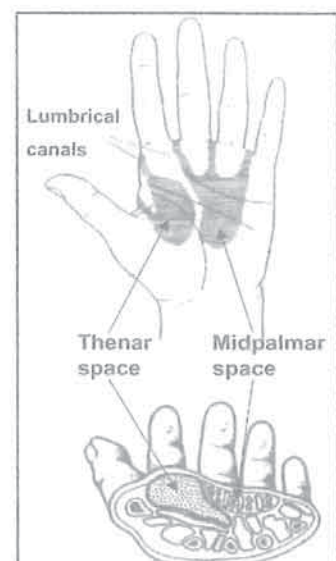


Fig. 17.4. Fascial spaces of the hand.

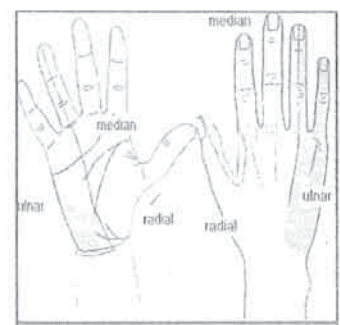


Fig. (17.5): Cutaneous innervation of the hand

Arteries

The radial and ulnar arteries communicate through the superficial and deep palmar arches. These arches give off digital branches that run on each side of the fingers, in a dorsal position to the corresponding digital nerves. Because of the free communication between the radial and ulnar arteries, ligation or block of one of them does not affect the viability of the hand.

Skin

The palmar skin is devoid of hair and sebaceous glands. It is tough and adherent to the underlying palmar aponeurosis.

The dorsal skin is soft and rests on loose areolar tissue. Because of this difference, oedema fluid tends to collect on the dorsum.

Hand infections

Overview

Aetiology. Infections of the hand are particularly prevalent among manual workers and house wives. In 90% of cases the offending organism is *Staphylococcus aureus*. The organism usually gets access to the hand tissues through minor injuries and punctures. If these are inadequately managed, infection sets in, with rapid development of suppurative, lymphangitis, and lymphadenitis.

Clinical features. In general, the condition presents with pain, swelling, and fever. The site of infection is known by finding the point of maximum tenderness rather than the area of oedema.

Investigations. Plain X-ray is needed if the presence of a foreign body is suspected. Blood sugar testing, for those with recurrent infections, may reveal the presence of diabetes mellitus.

Treatment. Once the condition is diagnosed, certain principles apply to the treatment of the different types of hand infection

1. **Antibiotic** administration is immediately started. The chosen drug should be effective against *Staph. aureus*, e.g., flucloxacillin, erythromycin, amoxycillin clavulanic acid combination, and first or second generation cephalosporins.
2. **Hand elevation** reduces pain and oedema. If there is marked swelling, the hand is bandaged in the position of function (Fig. 17.6). The metacarpo-phalangeal joints are flexed while the interphalangeal joints are extended. The wrist is slightly extended.
3. **Surgical drainage** is indicated if pus formation is evident from the start, or if there is no response to one day intensive antibiotic therapy.



Fig. 17.6. Immobilization in position of function.

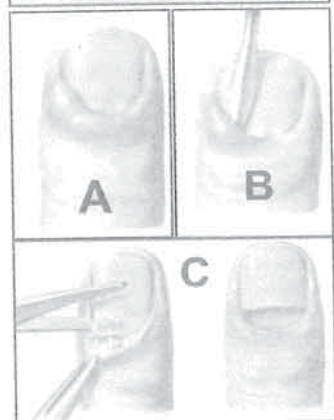


Fig. 17.7. Acute paronychia. A. Appearance. B. Drainage of localized paronychia. C. Drainage of cases with subungual extension.

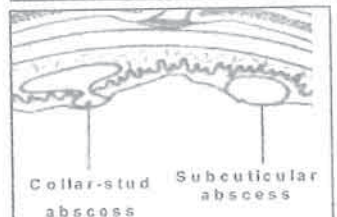


Fig. 17.8. Subcuticular and subcutaneous pus collections. The latter has formed a collar-stud abscess.



Fig. 17.9. Distal pulp space infection and its drainage.

- a. Acute paronychia and distal pulp space infection can be drained using local ring anaesthesia (without adrenaline) at the root of the finger. Otherwise, general anaesthesia is preferred.
 - b. To obtain a clear bloodless field, a pneumatic tourniquet is applied around the upper arm, the hand is elevated for a few minutes and then the tourniquet is inflated.
 - c. Appropriate skin incisions are used, and pus is drained by a sinus forceps.
 - d. Pus is sampled for culture and sensitivity.
 - e. Soft rubber drains are preferred, e.g. a piece of surgical glove.
4. **Postoperative elevation**, physiotherapy, and wound dressings.

Classification

1. **Cutaneous** and subcutaneous infections.
 - a. Paronychia.
 - b. Subcuticular and subcutaneous whitlow.
 - c. Pulp space infection.
 - d. Web space infection,
2. **Fascial spaces** infection.
 - a. Midpalmar space infection.
 - b. Thenar space infection.
 - c. Hypothenar space infection.
 - d. Space of Parona infection.
3. **Synovial sheaths** infection.
 - a. Acute digital tenosynovitis.
 - b. Ulnar bursitis.
 - c. Radial bursitis.
4. **Bone** and joint infections.

Paronychia

Acute paronychia. Acute suppurative infection of the nail fold is the commonest form of hand infections. Pus can be drained using a fine tipped scalpel to raise the nail fold and to incise the skin cap through which pus points. If there is a subungual extension, the related part of the nail is excised to provide proper drainage (Fig. 17.7).

Chronic paronychia, on the other hand, develops in the chronically wet nails of dishwashers. Associated fungal infection and nail deformities are common. The condition is treated by keeping the hands dry, and with antifungal therapy. Removal of the nail is commonly required.

Subcuticular and subcutaneous whitlow

Subepithelial collection of pus is called subcuticular whitlow. Drainage is afforded by excision of the insensitive roof without anaesthesia.

If this roof is callous, pus may trickle down through the dermis forming another subdermal component. The result is a 'collar-stud' abscess, which requires anaesthesia for its evacuation.

A subcutaneous abscess may also form without a subcuticular component.

Pulp space infection

The distal pulp space is the one that is commonly infected.

Anatomy. This space lies anterior to the distal phalanx and contains fatty tissue that is divided into locules by multiple septa running from the skin to the front of the bone. The space is closed proximally by the insertion of the profundus tendon to the palmar surface of the base of the distal phalanx. The digital artery divides in the middle segment of the finger into an epiphyseal branch which enters the articular end outside the pulp and a diaphyseal branch which traverses the pulp.

Clinical features and complications. In view of the division of this space into tight compartments, infection with its accompanying oedema produce a high rise of tissue pressure. This results in severe pain, and if neglected, the likelihood of affection of the digital vessels with septic thrombophlebitis; thus producing osteomyelitis of the shaft of the terminal phalanx. Fortunately complete regeneration can be obtained from the undamaged proximal part which is supplied by the epiphyseal branch.



Treatment. Early surgical drainage is indicated, the incision is sited directly over the most tender point (Fig. 17.9). An important dictum to remember is "Don't wait for fluctuation in cases of pulp space infection".

Fig. 17. 10. Web space infection.

Web space infection

Infection of a web space produces a swelling that leads to separation of the two adjacent fingers (Fig. 17.10). Spread may occur along the lumbricals to involve the midpalmar space. Pus is evacuated through a dorsal longitudinal incision between the fingers.



Midpalmar space infection

Infection occurs deep to the tough palmar aponeurosis either from punctures or from spread of either web space or tendon sheath infection (Fig. 17.11).



The condition is characterized by marked dorsal oedema. Pus is evacuated through an incision in one of the transverse palmar creases. To avoid injury of deep important structures, the incision is made through the skin only and then a sinus forceps is thrust inwards and is gently opened to let out pus (Hilton's method).

Thenar and hypothenar spaces infection

The thenar space is bounded anteriorly by the palmar fascia and flexor tendons of the index and thumb, posteriorly by the adductor pollicis and medially by the fibrous septum of the palm. Clinically infection of the thenar space presents by ballooning of the thenar eminence and oedema of the dorsum of the hand.

Fig. 17. 11. Midpalmar space infection. In this case it is an extension from tenosynovitis. Notice the marked dorsal oedema.

This infection is drained through an incision at the site of maximum tenderness or where pus points at the skin. An incision that is done along the medial side of the thenar

eminence should stop 2 cm distal to the distal wrist crease to avoid injury of the motor branch of the median nerve that supplies the thenar muscles.

Infection of the space of Parona

This space lies in the distal part of the forearm between the pronator quadratus and the flexor muscles. Drainage should be along the ulnar side of the forearm.

Acute tenosynovitis

This is the most serious of hand infections.

Aetiology. It is usually the sequel of deep pin pricks.

Clinical features. Involvement of a digital flexor sheath produces pain and swelling of the finger which the patient keeps in the semiflexed position. Any attempt at active or passive movement stretches the inflamed synovium and induces severe pain (Fig. 17.13). Tenderness is maximum just proximal to the crease over the metacarpophalangeal joints, which is the proximal extent of the synovial sac.

Little finger sheath affection is likely to spread to the ulnar bursa producing marked hand swelling, semiflexion and limitation of movement of the medial four fingers, and diffuse palm tenderness that may extend to the distal part of the forearm. The maximum point of tenderness may be on the ulnar side of the palm between the two palmar creases (Kanavel's sign). Radial bursitis produces a similar picture in the thumb.

Complications. Acute tenosynovitis is likely to result in complications.

1. Thrombo-phlebitis of the tendon vessels that run along the vinculae leads to its sloughing. The end result is a stiff useless finger.
2. Infection may also spread to the midpalmar space and the space of Parona.

Treatment

Lack of response to intensive conservative treatment is an indication for surgery. The inflamed synovial sheath is drained through two incisions, one at its proximal and another at its distal end. A fine catheter is inserted through each incision to allow continuous drainage and irrigation of the sheath with an antibiotic solution.

Ingrowing toe nail

This is a common problem in which a sharp edge of the big toe nail impinges on the adjacent skin fold. Young males predominate.

Aetiology

The predisposing factors are



Fig. 17.12. Thenar space infection and its drainage.

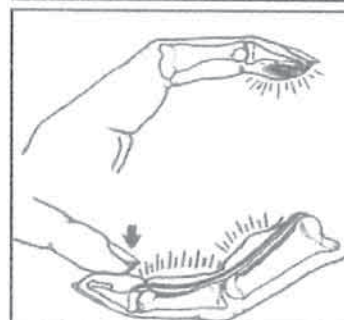


Fig. 17.13. Manifestations of flexor tenosynovitis are: 1. Slight flexion 2. Swelling 3. Tenderness over flexor tendon sheath 4. Pain on passive extension.



Fig. 17.14. Pathogenesis of ingrowing toe nail.

1. Faulty nail trimming. Oblique trimming of the nail sides may leave behind a sharp spike that starts the condition (Fig. 17.14).
2. Wearing tight shoes further thrusts this spike in the soft tissues.

Complications

Infection commonly sets in, and the condition may be made worse by attempts of the patient to cut away the nail.

Treatment

Conservative treatment is indicated in early cases. The aim is to lift off the nail spike (Fig. 17.15).

- A pledget of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail to raise it up and to ease away the injured tissues.
- Thinning the centre of the nail makes it pliable and facilitates elevation of the edge. The pledget is changed daily until the nail grows past the nail fold.
- The patient is instructed to avoid tight shoes, and is shown the proper technique of square nail trimming where the centre of the nail is kept at the same level or even shorter than its edges.

Surgery is indicated for failure of conservative treatment, and for suppurative cases.

Simple avulsion of the nail provides rapid relief of severe suppurative infection, but recurrence is common with regrowth of the nail. Wedge (segmental) excision of the germinal matrix is the definitive treatment (Fig. 17.16).

The nail grows from a germinal matrix that shows as a white crescent (lunula) under its proximal part. Removal of the lateral or medial part of the germinal matrix prevents regrowth of the corresponding part of the nail.

1. The operation is done under general or local anaesthesia.
2. A tourniquet is desirable to provide a clear bloodless field.
3. The side of the nail that is embedded is excised together with the infected skin and granulation tissue.
4. The tough white germinal matrix is excised back to its origin, just distal to the interphalangeal joint.
5. Phenol application may be added to ensure complete ablation of this part of the germinal matrix.
6. If the area is heavily infected, the wound is left open to granulate, otherwise its edges are loosely approximated.

Hand injuries

Classification

1. Tidy injuries. These are caused by sharp objects as knives and broken glass (Fig. 17.17A). The skin is cleanly cut and the tendons, nerves and vessels are commonly affected.

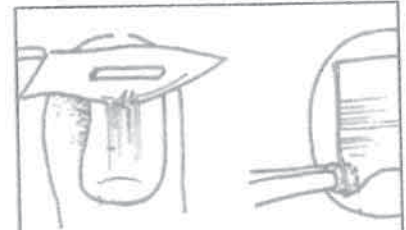


Fig. 17.15. Conservative treatment of ingrowing toe nail.



Fig. 17.16. Surgery for ingrowing toe nail.

2. **Untidy injuries.** These are caused by crushing forces and by burns and cause tissue devitalization (Fig. 17.17B). All hand tissues may be affected including bones.

Emergency care

The priorities of treatment of the multiple injury patient are outlined in chapter 2. An amputated hand or digit is kept in a clean polythene bag and is placed in a container full of cold water at 4°C (it should not be frozen). The patient is then urgently referred to a centre that is equipped with microsurgery facilities. Prophylactic antibiotics and tetanus prophylaxis are administered when indicated.

Assessment of injury

Clinical and radiological assessment should include the skin, nerves, arteries, tendons, and skeleton. The wound is inspected and the hand is examined for deformities, and for sensory and motor deficits. In many cases pain limits the value of these tests, and the final diagnosis of the extent of injury is obtained after surgical exploration.



Fig. 17.17.
A Clean cut.
B Crush injury.

Treatment of minor injuries

- A subungual haematoma is a painful condition (Fig. 17.18). Relief of pain is obtained by evacuating the blood accumulating beneath the nail. A hole is drilled in the nail using the needle of a syringe. The procedure is painless and, therefore, requires no anaesthesia.
- A minor skin wound is stitched under local infiltration anaesthesia.



Fig. 17.18. Subungual haematoma.

Principles of surgery for hand injuries

1. **Anaesthesia.** General anaesthesia is preferred. Alternatives are local intravenous anaesthesia with a tourniquet, and nerve blocks with local anaesthetics. The brachial plexus, median, or ulnar nerve may be blocked according to the site of injury. Digital nerve block is used for injuries of the distal parts of the fingers. It should be remembered that the digital arteries are end arteries, and if they are thrown into prolonged spasm by the addition of adrenaline to the digital block, gangrene of the finger may occur.
2. **A tourniquet** provides a clear bloodless field. It should be released within one hour. The use of magnifying loops or microscope is desirable,
3. **Careful wound toilet**, using saline irrigation, removes foreign bodies and debris.
4. **Excision of devitalized tissues** in untidy injuries.
5. **Haemostasis** is obtained by ligating small bleeding vessels. An injured radial or ulnar artery can be ligated without affecting viability of the hand. If facilities and experience are available, repair of an injured artery is a better alternative. Arterial repair is a necessity if both main arteries are injured.

6. **Nerves.** Primary repair of cut nerves is indicated in tidy wounds. For untidy injuries or evidently contaminated wounds, primary nerve repair is likely to fail. The repair is better postponed after wound healing and resolution of inflammation.
7. **Tendons.** The same principles of timing the repair apply to tendon injuries. Suturing is done with nonabsorbable nonirritant material, e.g. prolene. Success of tendon repair is endangered by the development of adhesions which limit its mobility. This problem is most marked in the case of flexor tendon injuries, in the area between the distal palmar crease and the middle of the fingers, where the flexor digitorum superficialis and profundus tendons are enclosed within the digital fibrous flexor sheath. With refinement of surgical technique, the tendency is now for direct suturing irrespective of the site of injury. Tendon grafting is reserved for complicated cases with loss of substance and for recurrent cases.
8. **Finger tip injuries with loss of skin.** If there is skin loss only, the finger tip is covered by split or full thickness graft. With finger tip amputations that expose the bone, skin grafts will not take, and local skin flaps are usually used. Examples of such solutions include cross finger flap, or the advancement of two lateral triangles of skin and subcutaneous tissue to cover the defect.
9. **Skin closure** is to be done without tension. Contaminated wounds are left open. Wounds of human and animal bites are highly contaminated with a mixture of bacteria, including anaerobes, and should never be sutured.
10. **Microsurgical replantation** of severed parts gives good results when it is done early for a clean cut amputation. The operation is useless if the part is severely crushed or if 12 hours have elapsed after the injury.

Postoperative care

- The hand is bandaged in the position of function, and is kept elevated for a few days to minimize oedema formation.
- Physiotherapy is started after one week to allow restoration of hand function.

Swellings of the hand

The great majority of hand swellings (94%) are benign.

Simple ganglion

Definition. This is a small cyst that contains a clear gelatinous fluid.

Pathogenesis. The ganglion forms as a result of protrusion of a joint or tendon synovial membrane that later becomes isolated to form a cyst full of synovial fluid. The fluid then thickens to form the characteristic jellylike ganglion content. Synovial membrane protrusion may be an acute or a chronic process, yet whatever the speed of development, a ganglion is most commonly found on the dorsum of the wrist.

Clinically a ganglion presents as a small, rounded cystic swelling usually on the dorsum of the wrist, less commonly on the dorsum of the foot. The swelling is usually painless. Sometimes, it is tense cystic giving a false impression of being hard.

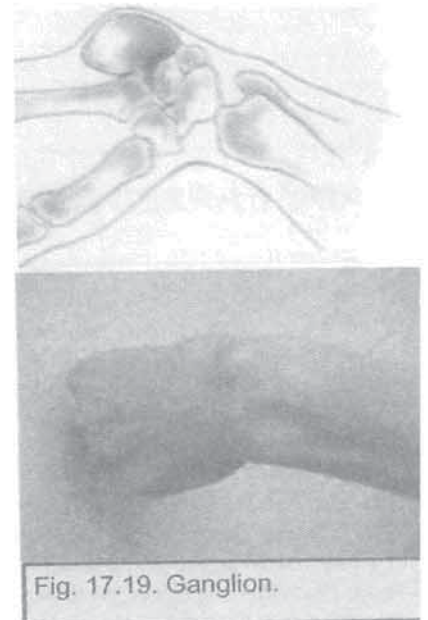


Fig. 17.19. Ganglion.

characteristic physical sign is that the mobility of the swelling is markedly restricted by stretching or by contraction of the related tendons.

Treatment of a simple ganglion is not indicated unless the patient insists. Aspiration, or rupturing the cyst by applying direct pressure leads to temporary disappearance followed by recurrent swelling. Excision is the only definitive treatment. This should be done under general anaesthesia and a tourniquet is applied. The wall should be excised completely, otherwise recurrence will occur.

Giant cell tumour of tendon sheath

The tumour forms a firm, painless mass on the volar or dorsal aspects of the fingers. The patient may present on account of its appearance. The tumour may also interfere with tendon movement, compress digital nerves, and erode bone. Treatment is by meticulous excision. Recurrence is treated by re-excision, combined with radiotherapy.

Glomus tumour

This is a tumour of the heat regulating arteriovenous shunts and the unmyelinated nerves that control them. It may occur anywhere in the skin, but is commonly found beneath a nail. The tumour is markedly painful and tender, and has a red or bluish colour. Glomus tumour must be differentiated from a subungual haematoma, exostosis, and melanoma. Treatment is by total excision under magnification.

Implantation dermoid cyst

Puncture wounds may drive in small pieces of skin in the subdermal tissues. The skin produces sebaceous material forming a cyst. Dermoid cysts are commonly present in the distal phalanx.

SURGERY OF NERVES

A nerve trunk is formed of collection of nerve fibres arranged in bundles bound together by connective tissue (Fig. 18.1).

- The whole nerve trunk is surrounded by a connective tissue fascia called epineurium.
- Each bundle of nerve fibres is surrounded by connective tissue called perineurium.
- The connective tissue around individual nerve fibres is called endoneurium.
- Each nerve fibre consists of the central axis cylinder surrounded by the myelin and the neurolemmal sheaths.

The fibres contained in a peripheral nerve may be motor, sensory, vasomotor or sudomotor. Unlike the central nervous system where divided tracts do not regenerate, injured peripheral nerves may recover to varying extent, depending on the severity of trauma.

CHAPTER CONTENTS

- Naive injuries — general considerations
- Cranial nerve injuries
- Spinal nerve injuries
- Sciatica
- Autonomic nervous system
- Nerve turnouts

Pathological types of nerve injuries

- **Neurapraxia** Functional block of nerves
- **Axonotmesis** Anatomic disruption of axon with no disruption of connective tissue.
- **Neurotmesis** Anatomic disruption of axon and connective tissue.

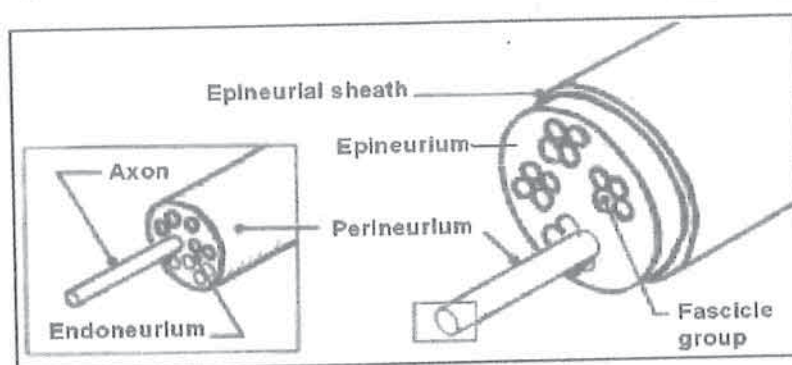


Fig. 18.1. Histology of a peripheral nerve.

Nerve injuries—general considerations

Aetiology

1. Open injuries. The nerve may be divided or lacerated in open wounds or may be damaged in crushes and burns.
2. Closed injuries. The nerve may be damaged by:
 - Contusion by sudden direct violence.
 - Compression against the adjacent bone by tourniquets, splints, plasters or crutches.
 - Traction (stretching) such as tearing of the lateral popliteal nerve in forcible adduction of the knee or tearing of the roots of the brachial plexus in birth injuries.
 - Fractures and dislocations. The nerve may be contused, compressed, stretched or rarely, lacerated by the displaced bone.
 - Ischaemia from damage or occlusion of the main artery.
 - Accidental injection of irritant substances as in the case of the sciatic and radial nerves.

Pathology

Nerve injuries are classified according to the extent of damage to the nerve fibres and sheath into 3 types

- **Neurapraxia** is a functional paralysis of conduction but the nerve is anatomically intact without any organic rupture.
 - It may be produced by minor traction or compression injuries or by the concussion and vibratory effect of a high velocity missile passing near the nerve.
 - The fibres remain intact within their sheaths and no degeneration of the axons occurs.
 - Clinically there is complete motor paralysis in the distribution of the nerve with a patchy loss of sensation or even with no sensory disturbance at all. The electrical excitability of the paralysed muscles remains normal.
 - Recovery takes place spontaneously in a matter of days or weeks.
- **Axonotmesis** is a partial or complete intrathecal rupture of the nerve fibres within an intact sheath.
 - It may be produced by contusion and traction injuries or may complicate fractures and dislocations.
 - The nerve fibres are damaged and Wallerian degeneration occurs in the distal portions of the torn axons, resulting in complete motor and sensory paralysis.
 - Subsequent regeneration of the axons occurs across the site of the damage and continues distally to the motor and sensory nerve endings. Recovery is, however, delayed until the growing axons reach their appropriate endings, and occurs first in the muscle groups nearest to the site of injury and lastly in the peripheral skin areas. The length of time required for the recovery depends upon the level of the lesion. After an initial delay of about 10 days, the axons proceed distally at a rate of approximately 1 mm per day and on arrival at their endings there is a further delay of 3 weeks before the end organs become activated. Recovery after such lesions is usually complete because the axons remain in their respective neurolemmal tubes.
- **Neurotmesis.** This term includes all forms of partial or complete anatomical division of the nerve.
 - Degenerative changes occur in the nerve, as in axonotmesis but the gap fills with a mass of fibrous tissue and regenerating axons known as bulb neuroma. Partial lesions produce a lateral or central neuroma. Complete division produces a terminal neuroma on the end of the proximal segment of the nerve, separated by an interval from the atrophied distal end. In the distal segment of the divided nerve Wallerian degeneration occurs.
 - The regenerating proximal axons cannot enter the distal neurolemmal tubes as they are blocked by the neuroma. Even if an axon can pass distally it is unlikely to go through the correct neurolemmal tube because of loss of alignment. A motor axon may go into a sensory distal tube or through a tube of a different muscle. Spontaneous recovery is, therefore impossible. After surgical repair recovery is poorest in mixed nerves and in motor nerves which supply a large number of small muscles because of maldistribution of the fibres. The quality of recovery is best in purely motor nerves which supply a few groups of large muscles such as the radial, and is worst in mixed nerves supplying a large number of muscles concerned with fine movement such as the ulnar and median nerves.

Clinical features

▪ Motor effects

- Deformity is often characteristic such as the claw hand deformity of ulnar paralysis.
- Loss of voluntary contraction of supplied muscles.
- Muscle atrophy. A few months after injury the paralysed muscles shrink, lose their cross striation and ultimately disappear to be completely replaced by fibrofatty tissue.
- Loss of reflexes. Every reflex, superficial or deep, whose arc crosses the point of injury on the nerve, is lost.

▪ Sensory effects

- Anaesthesia. Immediately after injury there is loss of sensation over the supplied area. Owing to the overlap of dermatomes, however, the area of anaesthesia is always less than that of the anatomical distribution of the nerve.
- Pain. Pain referred to the area of cutaneous distribution of an injured nerve is more common in partial than in complete lesions and is a favourable sign. Severe burning pain is a rare complication of nerve injuries and is known as causalgia.

- **Sudomotor effects.** After a complete injury sweating ceases in the denervated part (anhydrosis), usually over a larger area than the area of anaesthesia.

- **Vasomotor effects.** Vasodilatation results from paralysis of the vasomotor fibres and the denervated skin becomes red and warm. After about 3 weeks, however, the denervated area becomes blue and cold, probably from loss of the afferent limb of the vasomotor reflex.

- **Trophic changes.** These probably result from disuse, sensory loss and vascular changes.

- The skin loses its pits and wrinkles and becomes smooth, thin and inelastic. Normal resistance to trauma is lost and ulceration occurs, especially in the fingers, toes, sole and heel. The nails become distorted and brittle. Loss of hair occurs in the denervated areas. There is loss of subcutaneous fat and the tips of the fingers may shrink and taper.
- Progressive muscles atrophy. The non-paralysed muscles and their tendons shorten and deformities become permanent by contraction of the fasciae and joint capsules.
- Contracture and ankylosis of joints develop more readily in painful than in painless lesions.
- The bones undergo progressive decalcification which is most marked in the proximal and distal extremities of the phalanges.

Diagnosis

In open injuries there is usually neurotemesis while in closed injuries axonotemesis is the usual insult.

Tinnel's sign. Tapping the nerve will initiate tingling sensation at the site of growing nerve fibrils. This sign is valuable in the follow-up of regeneration of closed nerve injuries or after nerve repair.

Investigations

1. **Nerve conduction velocity (NCV).** The idea of this test is to measure the time that elapses after stimulation of a nerve at a chosen point to induce a muscle action potential. Normal NCV for most nerves is 50-70 m/sec. Neurapraxia does not

interfere with NCV. However, after complete transection of a nerve, NCV progressively decreases and becomes 20-40% of normal after one month. NCV is most suitable for the diagnosis of compression neuropathy and is very useful in detecting regenerating axons as NCV will gradually increase.

2. **Electromyography.** This is done by inserting a needle in a muscle supplied by the nerve to be studied. Normally at rest there is no electrical activity recorded by the needle electrode, but when the muscle voluntarily contracts an electrical activity of specific pattern is recorded. Two to four weeks after complete nerve injury, the denervated muscle shows spontaneous fibrillations. These fibrillations remain so long as the muscle is still viable, but if complete fibrosis occurs, no electrical activity is detected and the muscle is no longer suitable for reinnervation.

Treatment

Conservative treatment

Closed injuries are more likely to be of the neurapraxia or axonotmesis types, both of which recover spontaneously. The aim of treatment is to prevent muscle stretching, minimize muscle wasting and preserve joint mobility, until spontaneous reinnervation occurs.

1. Splintage is essential to prevent overstretching of the paralysed muscles but it should permit free movement of the unparalysed muscles. The splint should be removed at least once a day to permit all the joints to be put through a full range of passive movement.
2. Physiotherapy. Active exercises should be supplemented by passive exercises and gentle massage to maintain the nutrition of the tissues and prevent oedema and joint stiffness.
3. Electrotherapy. Stimulation of the paralysed muscles with a galvanic current is essential to minimize the wasting and fibrosis which occur during the period of denervation. It should be started early and at least 50 strong contractions of each paralysed muscle should be obtained every day.
4. Protection of the skin. The patient should be warned of the susceptibility of the anaesthetic skin to injury and heat.

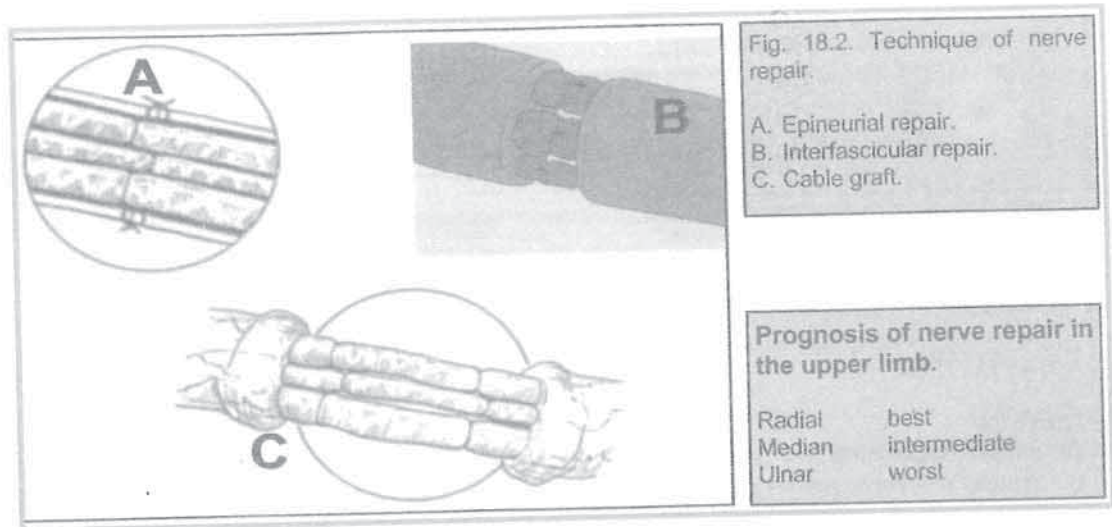
Surgical treatment

Indications

1. Open nerve injuries.
2. Closed nerve injuries if
 - a. Failure of recovery at a rate of 1 mm/day.
 - b. Failure of progress of Tinel's sign.
 - c. If there is a palpable neuroma.

Timing of surgery

1. **Primary nerve suture.** In clean incised wounds immediate primary nerve suture is the ideal treatment as it gives the best results.
2. **Secondary nerve suture.** In contaminated or lacerated wounds primary nerve suture should not be attempted. Secondary nerve suture is performed after complete healing of the skin. This policy is adopted because nerve suture needs adequate mobilization of both ends and opening new tissues planes that will allow spread of infection. In this case the aim at the primary operation should be to obtain closure of the skin and early healing of the wound. The ends of the divided nerve are loosely approximated with a single stitch of silk to preserve correct orientation of the fibres and prevent undue retraction.



Technique (Fig. 18.2)

1. General anaesthesia with a tourniquet.
2. A long incision.
3. Mobilization of the nerve ends.
4. Nerve conduction studies of surgically exposed nerves can provide direct evidence of the extent of neural injury
 - a. Lack of conduction across the injured segment or distally warrants excision and grafting of the injured segment.
 - b. Conduction across the injured segment warrants neurolysis.
5. Resection of the neuroma and the damaged parts of nerve ends using a sharp knife.
6. Nerve suture. One of two methods is used for accurate apposition of cut ends.
 - a. Epineurial repair. Interrupted 6/0 prolene sutures pick up the nerve sheath only (epineurium).
 - b. Interfascicular repair is a more accurate method which is performed using the operating microscope. The fasciculi of the proximal and distal segments are very accurately opposed by passing the sutures through the perineurium.
7. Nerve grafting. If the defect could not be easily approximated after previous procedures, nerve grafting should be done. A small cutaneous nerve is harvested; usually the sural nerve or the ilioinguinal nerve. This nerve is divided into segments that match the size of the defect. This type of multiple nerve grafts (2-4) bridging the defect looks like a cable hence the name "cable grafting". For successful take of the cable graft, a well vascularized bed should be available.

Results

The prospects of success depend on several factors.

1. The injured nerve. The radial nerve is by far the most satisfactory because it contains a predominance of motor fibres and there is less chance of maldistribution of the fibres during regeneration.
2. Level of suture. With a high-level injury the length of time taken by the regenerating fibres to reach the more distal muscles reduces the chance of a satisfactory recovery.

3. Time interval between injury and suture. The earlier the repair, the better is the result since a long period of denervation of a muscle allows more wasting and degeneration of the muscle fibres. A muscle which has been deprived of its nerve supply for more than 2 years cannot be expected to show any degree of recovery owing to irreversible changes in the motor end plates.
4. Extent of injury. Large gaps between cut ends have a bad prognosis.
5. Age and general condition of the patient. The results of nerve suture are particularly good in children and adolescents.

Secondary treatment

A number of operations are available to improve function when recovery of the nerve is impossible. These include

1. Arthrodesis, e.g., arthrodesis of the shoulder for paralysis of the abductors and arthrodesis of the tarsal joints for paralytic talipes.
2. Tendon transplantation, e.g., for radial palsy.
3. Amputation, e.g., amputation of the arm for total brachial plexus injuries and amputation of the leg for sciatic lesions.

Cranial nerve iniuries

Cranial nerve palsies are discussed in detail in medical textbooks. Only the facial, accessory and hypoglossal nerves are discussed here.

Facial nerve

Aetiology

The seventh cranial nerve may be involved by a variety of lesions.

- **Intracranial lesions** may be supranuclear, nuclear or infranuclear, the nucleus of the nerve being in the floor of the fourth ventricle.
 - Supranuclear lesions produce an upper motor neurone paralysis of the lower half of the opposite side of the face only since the occipitofrontalis and orbicularis oculi muscles enjoy a bilateral innervation.
 - In nuclear lesions the whole face and the sixth nerve on the same side are affected with rapid atrophy of the facial muscles, together with crossed hemiplegia, since the motor decussation takes place at a lower level.
 - An infranuclear lesion occasionally results from pressure of a tumour in the cerebellopontine angle on the root of the nerve and gives rise to paralysis of the same side of the face together with deafness and cerebellar signs.
- **Cranial lesions.** The intraosseous portion of the nerve may be damaged in fractures of the base of the skull, middle ear disease or operations on the mastoid antrum. In this part of its course any lesion which affects the nerve is liable also to involve the auditory nerve, the petrosal nerves or the chorda tympani.
- **Extracranial lesions**
 - The nerve itself is commonly involved by Bell's palsy. This is probably due to herpetic neuritis of the nerve and may follow exposure to cold or an air draught. Swelling within the sheath of the nerve extends into the stylomastoid foramen and so the nerve is compressed within its bony canal. Absorption of the exudate usually occurs before the pressure has damaged the nerve permanently, but in about 10% of cases some degree of paralysis persists.
 - The nerve trunk or branches may be damaged by injuries, operations or malignant tumours in the parotid region.

Clinical features

- **Face.** The affected side of the face is flat, motionless and expressionless. On looking upwards the corrugations of the forehead are not apparent. On attempting to move the face, as in laughing or showing the teeth, the muscles on the non-paralysed side alone are contracted. Marked asymmetry results from the drawing over of the opposite side,
- **Eye.** The eye cannot be closed, and on attempting to do so the eyeball rolls upwards and outwards. Epiphora results from drooping and relaxation of the lower eyelid and corneal ulceration may follow due to exposure.
- **Mouth.** The lips cannot be closed firmly and so whistling and blowing cannot be performed. Food collects between the cheek and teeth from paralysis of the buccinator and the patient, after a meal, has to clean out the debris with his fingers.
- **Tongue.** Loss of taste in the anterior two-thirds of the tongue indicates a lesion proximal to the entrance of the chorda tympani (about 6 mm above the stylomastoid foramen).

Treatment

- **Conservative** treatment for Bell's palsy Electrical treatment and massage are prescribed to maintain health of the muscles and prevent atrophy. During recovery, active facial movements in front of a mirror should be practised several times daily. Most cases of Bell's palsy begin to recover after 3-4 weeks and recovery is complete in 3-6 months.
- **Operative** treatment for injuries
 - Nerve suture should be performed if the nerve is accidentally divided. If a nerve graft is needed to bridge a defect in the facial nerve, the easiest source is the great auricular nerve as it lies within the area of the parotidectomy operation which is a common cause of injury.
 - If repair is impossible hypoglossal anastomosis may be resorted to. The hypoglossal nerve is intentionally divided and its proximal end is anastomosed to the distal end of the injured facial nerve.
 - In hopeless cases, asymmetry of the face may be improved by a plastic operation such as the insertion of fascial strips to sling up the corner of the mouth to the zygoma.

Accessory nerve

- The eleventh nerve may be injured by fractures of the posterior fossa involving the jugular foramen but more commonly it is damaged during operations on the neck particularly for removal of tuberculous or malignant lymph nodes.
- Division of the nerve in the anterior triangle results only in partial paralysis of the sternomastoid and trapezius muscles since they receive an additional nerve supply from the cervical plexus.
- If the nerve is injured in the posterior triangle the trapezius alone is affected. Paralysis of the trapezius causes drooping of the shoulder, tilting of the scapula and inability to abduct the arm above the right angle. Weakness of the whole arm results from imperfect fixation of the scapula.

Hypoglossal nerve

- This nerve may be injured during operations for removal of tuberculous lymph nodes and may be involved by malignant diseases. It usually escapes, however, fractures of the base of the skull, since the anterior condyloid foramen is protected by a bony ridge which deflects the fracture towards the foramen magnum.

- Paralysis of the nerve is followed by hemiatrophy of the tongue which becomes directed towards the paralysed side on protrusion.

Spinal nerve injuries

Brachial plexus

Aetiology

1. **Open injuries.** The plexus may be injured in gunshot wounds and stabs in the lower part of the posterior triangle but such injuries are rare.
2. **Traction injuries** may arise from
 - a. Forcible depression of the shoulder as when heavy weights fall on the shoulder. Long-continued depression of the shoulders may also cause undue traction upon the roots of the plexus, especially of the fifth and sixth nerves.
 - b. Hyperabduction of the arm as when the body is dragged along the ground by the arm or when a person clutches at some support when falling. The damage falls chiefly on the eighth cervical and first thoracic nerves.
 - c. Birth-injury may occur equally in vertex or breech presentations from forcible stretching of the head during delivery.
3. **Pressure injuries.** Fractures of the clavicle and dislocations of the shoulder or attempts to reduce them may result in injury to the plexus, especially to the medial cord.

Clinical features

Three types of lesions are encountered.

1. **Whole plexus type.** This type is rare since an injury sufficiently severe to damage all the roots of the plexus is liable to inflict fatal injuries on adjacent important structures. In such complete lesions the whole arm is paralysed and lies flaccid and anaesthetic by the patient's side. Sensation is present only on the medial side of the arm as far down as the elbow (intercostobrachial nerve) and for a more limited area on the outer side (supraclavicular nerves). There may be sympathetic paralysis of the eye and face producing ptosis, myosis, enophthalmos and anhidrosis of one side of the face (Homer's syndrome).
2. **Upper-arm type (Erb-Duchenne paralysis).** The fifth and sometimes the sixth cervical nerves are involved
 - a. The limb hangs by the side with the forearm extended and pronated in the so-called "policeman tip" or "waiter's tip position" (Fig. 18.3) and if the sixth nerve escapes the arm will be internally rotated by the unopposed subscapularis. The arm cannot be abducted or rotated, the elbow cannot be flexed and the forearm cannot be supinated, but the movements of the hand and wrist are unimpaired. Sensory changes are absent if the fifth nerve only is involved, but if the sixth nerve also suffers, an area of anaesthesia is present over the outer side of the arm.
3. **Lower-arm type (Klumpke's paralysis).** The first thoracic and sometimes the eighth cervical nerves are affected.
 - a. There is paralysis of the flexors of the wrist and fingers (8th cervical) and of the intrinsic muscles of the hand (first thoracic).

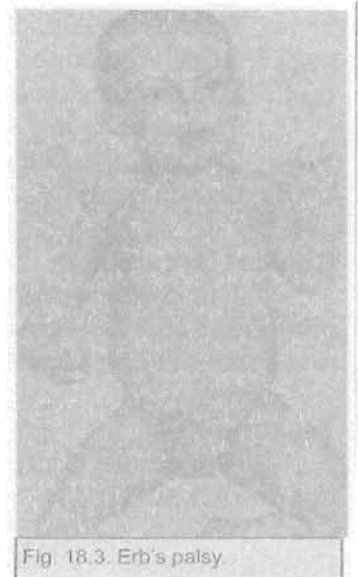


Fig. 18.3. Erb's palsy.

- b. A claw-hand results with wasting of all small muscles of the hand.
- c. Anaesthesia is present along the inner side of the forearm and the inner three and half fingers (ulnar distribution).
- d. Horner's syndrome may occur if the sympathetic fibres are involved. It indicates a high lesion and a bad prognosis.

Treatment

Conservative treatment

In closed injuries the lesion is usually a mixed one due to a combination of neurapraxia, axonotmesis and neurotmesis and so conservative treatment is often followed by progressive improvement.

1. The arm is splinted in right-angled abduction to relax the deltoid and spinati, the forearm being flexed and supinated to relax the biceps, brachialis and supinators.
2. Oedema is controlled by elevation and massage.
3. Stiffness is prevented by active and passive movements of all joints.
4. Electrotherapy is employed to maintain the tone and nutrition of the paralysed muscles.

Neurapraxia recovers within 3 or 4 weeks but axonotmesis takes a very long time and physiotherapy should be continued for 2 years.

Surgical treatment

Exploration of the plexus is indicated in degenerative lesions which fail to show any evidence of recovery within 2 months, particularly when a palpable neuroma develops. Scar tissue is removed and the nerve ends are freshened, identified and sutured together. The operation is difficult and the results are often disappointing.

Circumflex nerve

Aetiology

This nerve is occasionally injured where it winds round the neck of the humerus by

1. Gunshot wounds.
2. Direct blows on the shoulder.
3. Fractures of the surgical neck of the humerus.
4. Dislocations of the shoulder.
5. Crutch palsy.

Clinical features

The deltoid muscle is paralyzed and wastes rapidly so that the acromion becomes prominent and the shoulder flattened. The patient is unable to raise the arm from the side, and there may be temporary anaesthesia over the posterior fold of the axilla.

Treatment

Any cause of pressure is removed and splintage, massage and electric stimulation are carried out.

Radial nerve

Aetiology

1. Injury in the axilla may result from pressure by crutches or may follow fractures and dislocations of the upper end of the humerus or attempts at their reduction. Crutch palsy can be avoided by the use of modern crutches of proper length provided with hand-grips.
2. Injury in the spiral groove may be due to:

- a. Fractures of the shaft of the humerus,
- b. Falling asleep with the arm lying across the edge of a chair or table (Saturday-night paralysis).
- c. Prolonged application of a tourniquet.
- d. Operations in which the outstretched arm has rested on the edge of the table.
- e. Intramuscular injections of irritant drugs.

Clinical features

Motor

There is paralysis of the following muscles

1. Triceps and anconeus. The forearm can only be extended by its own weight. They escape in injuries below the axilla.
2. Supinator and brachioradialis. The hand is pronated but supination can still be performed by the biceps.
3. Extensors of the wrist. Wrist-drop (Fig. 18.4) is present and the grasp is weak since the flexors cannot be tightened by extending the wrist joint.
4. Extensors of the fingers and thumb which hang limp and motionless and are sometimes flexed into the palm by the unopposed action of the flexor muscles. If, however, the wrist and proximal phalanges are supported and extended, the middle and distal phalanges can be straightened by the action of the interossei and lumbricals (trick movement). To test, therefore, for extensor paralysis the metacarpophalangeal joints should be flexed to throw the interossei out of action.

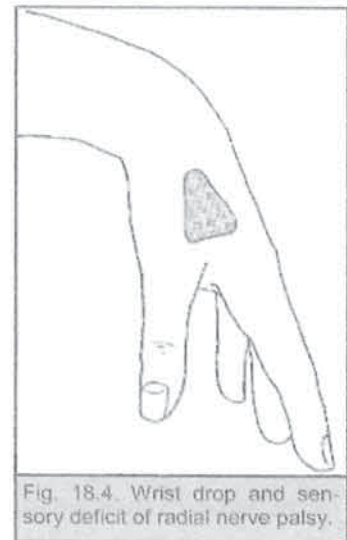


Fig. 18.4. Wrist drop and sensory deficit of radial nerve palsy.

Sensory

Anaesthesia is variable

- If the nerve is injured high up in the arm above the origin of its lateral cutaneous branch, loss of sensation over the radial two thirds of the dorsum of the hand is expected but practically the area of sensory loss is usually restricted to the first interosseous space dorsally (Fig. 18.4).
- A lesion in the upper third of forearm causes no sensory loss.

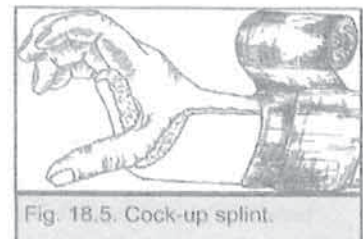


Fig. 18.5. Cock-up splint.

Treatment

1. Conservative treatment. Stretching of the paralysed muscles should be prevented by placing the hand in a position of slight hyperextension on a "cock-up" splint (Fig. 18.5). Massage of the forearm and electrical treatment are continued until recovery is complete.
2. Nerve repair. If no signs of recovery appear within 6 weeks operation is indicated. The prognosis of operations on this nerve is better than in the case of any other nerve in the body because it is almost purely motor and because it controls coarse and unskilled movements. After suture in the arm, recovery may be expected in 9-12 months.

3. Tendon transplantation. In cases where operation has failed, or where the posterior interosseous nerve has been damaged, the wrist-drop can be corrected and excellent function obtained by tendon transplantation. The tendon of the flexor carpi ulnaris is grafted into the extensors of the fingers, the tendon of the flexor carpi radialis into the extensor tendons of the thumb and the tendon of the pronator teres into the lower ends of the divided radial extensors of the carpus.

Median nerve

Aetiology

1. The nerve may be injured above the elbow by tourniquets, in which case other nerves, particularly the radial, are also involved.
2. It may be injured at the elbow by fractures of the lower end of the humerus or dislocations of the elbow joint.
3. It may be involved in gunshot wounds of the forearm.
4. The nerve, however, is most frequently injured just above the wrist by wounds from various causes, especially glass wounds due to bursting of bottles or thrusting the hand through a window.
5. Median nerve compression. See carpal tunnel syndrome (chapter 16).



Fig. 18.6. Pointing index of median nerve injury.

Clinical features

Motor

1. There is paralysis of the 2 pronators with loss of pronation of the forearm.
2. The flexor carpi radialis is paralysed causing defective flexion of the wrist on the radial side and impaired radial abduction.
3. The flexor pollicis longus, palmaris longus, flexor sublimis and outer half of the flexor profundus are paralysed, leading to loss of power in the hand grasp, especially on the radial side. The terminal phalanx of the thumb cannot be flexed and this is well demonstrated by asking the patient to clench the fist when the thumb cannot be flexed over the knuckles of the fingers. The index finger cannot be flexed at the interphalangeal joints or at the metacarpophalangeal joint due to the paralysis of its long two flexors and the lateral lumbrical muscle - "pointing index"- (Fig. 18.6) but flexion of the other fingers is performed by that portion of the flexor digitorum profundus which is supplied by the ulnar nerve.
4. The outer group of short muscles of the thumb- abductor pollicis brevis, opponens pollicis and flexor pollicis brevis-are paralysed. The thenar eminence is wasted and flattened (Fig. 18.7) and the thumb is extended by the side of the fingers with its metacarpal apparently on the same plane as the other metacarpals-the so-called "simian" or "ape-like hand". The patient cannot raise the thumb forwards at right angles to the plane of the palm (abduction) or to swing it forwards and inwards to touch the tip of the little finger (opposition) (Fig. 18.8).
5. There is paralysis of the outer 2 lumbricals.



Fig. 18.7. Wasting of thenar muscles.

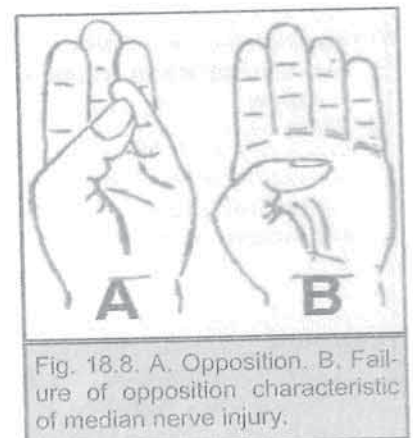


Fig. 18.8. A. Opposition. B. Failure of opposition characteristic of median nerve injury.

6. Injuries at the wrist are often associated with great impairment of mobility in the hand and fingers due to involvement of the tendons in scar tissue and the formation of adhesions in the joints and tendon sheaths but the disability is particularly severe if the tendons are also severed.

Sensory

Sensation is lost over the palmar aspect of the radial side of the hand, as well as of the thumb, index, middle and half the ring finger (Fig. 18.9).

Trophic

The tips of the fingers are shrunken and tapering and cigarette burns are often seen on the anaesthetic fingers. Severe causalgia may complicate partial injuries of the nerve.

Treatment

Operative treatment is often indicated to liberate the nerve and tendon from dense scar tissue and to suture the nerve if completely divided. The results are often unsatisfactory, but the operation should always be undertaken since a complete lesion of the nerve renders the hand almost completely useless.



Fig. 18.9. Sensory deficit of median nerve injury.

Ulnar nerve

Aetiology

1. Wounds, fractures and dislocations in the region of the elbow. Delayed ulnar neuritis often follows cubitus valgus deformity due to old injury of the lower end of the humerus.
2. Wounds at the wrist.

Clinical features

Motor

Division of the ulnar nerve is important by reason of the muscular rather than the sensory paralysis.

1. There is paralysis of the flexor carpi ulnaris, causing weakness in flexion and flattening of the inner border of the forearm.
2. The inner half of the flexor profundus is paralysed and the grasp of the hand is weakened, especially in the ring and little fingers. Flexion of the terminal phalanx of the ring and little finger is affected. Flexion of the terminal phalanx is tested by fixing the proximal interphalangeal joint and asking the patient to flex the terminal phalanx.
3. Paralysis of the small muscles of the hand also results, with the exception of the thenar muscles and the outer 2 lumbricals.
 - a. Paralysis of the medial two lumbricals causes flexion of the interphalangeal joints and hyperextension of the metacarpo phalangea joints of the medial two fingers (partial claw- hand, Fig. 18.10). In high ulnar nerve lesions the claw hand deformity is not very apparent due to a concomitant paralysis of the flexor digitorum profundus (ulnar paradox).



Fig. 18.10. Partial claw hand deformity of ulnar nerve injury. Wasting of hypothenar muscles is evident.

- b. Considerable wasting with flattening of the hypothenar eminence (Fig. 18.10) and hollowing of the interosseous spaces (Fig. 18.11) due to paralysis and atrophy of dorsal interossei.
- c. Inability to abduct and adduct the fingers due to weakness of the interosseous muscles. The patient cannot grip a sheet of paper between the extended fingers (card-board test, Fig. 18.12).
- d. Froment's sign. If the patient pinches a piece of paper between the thumb and fingers the terminal phalanx of the thumb assumes a flexed position since weakness of the adductor pollicis is compensated for by over action of the thumb flexors (Fig. 18.13).

Sensory

There is loss of sensation over the inner one and half fingers in front and behind, as well as over the ulnar side of the hand and wrist (Fig. 18.14). In lesions at the wrist sensation is lost only on the anterior aspect since the dorsal cutaneous branch of the ulnar leaves the main trunk about 2 inches above the styloid process of the ulna. Trophic changes are usually well-marked and correspond to the area of sensory loss.



Fig. 18.14. Sensory deficit of ulnar nerve injury.

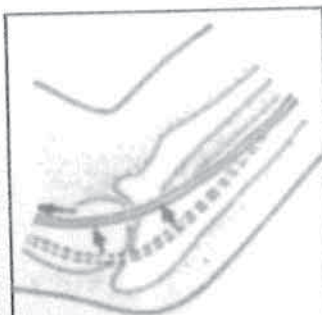


Fig. 18.15. Anterior interposition of ulnar nerve.



Fig. 18.16. Complete claw hand (combined ulnar & median nerve injury)



Fig. 18.11. Hollowing of interosseous spaces.

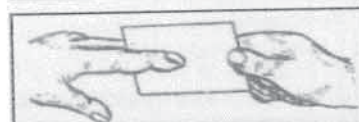


Fig. 18.12. Card board test.

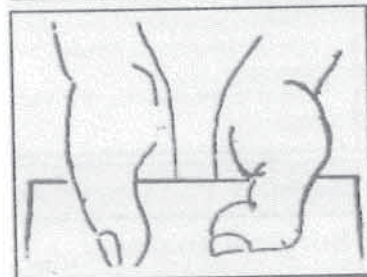


Fig. 18.13. Froment's test shows weakness of left adductor pollicis

Treatment

1. Nerve repair. If there is a gap, it can be shortened by transposition of the nerve to the front of the elbow. The prognosis is even less favourable than in the case of median nerve repair and the disability of a complete lesion is almost as great.
2. Anterior transposition of the nerve (Fig. 18.15) is indicated for
 - a. Delayed neuritis due to cubitus valgus deformity.
 - b. With nerve repair to reduce tension on the suture line.

Differential diagnosis of claw hand deformity

- Ulnar nerve injury.
- Lower brachial plexus injury.
- Dupuytren's contracture.
- Volkmann's ischaemic contracture.
- Post-burn contracture.

N.B. A cut wound at the front of the wrist may injure the ulnar and median nerves, one or more of the flexor tendons in addition to the blood vessels. Combined median and ulnar nerve injury produces complete claw hand deformity (Fig. 18.16).

Sciatic nerve

Aetiology

1. Deep wounds of the buttock and thigh.
2. Dislocations of the hip.
3. Fractures of the pelvis.
4. Intramuscular injection of drugs.

Clinical features

Motor

There is paralysis of all muscles below the knee and drop foot results. If the nerve is damaged near its exit from the pelvis, the hamstrings are paralysed but some degree of flexion of the knee is possible through the action of the sartorius and gracilis muscles.

Sensory

There is anaesthesia of the leg and foot with the exception of the area supplied by the saphenous nerve along the medial aspect of both. Trophic changes are common on the sole of the foot and to, and if the nerve lesion is partial severe pain (causalgia) may be experienced in the sole.

Treatment

1. Conservative treatment follows the general rules laid down before. The deformity is controlled by wearing a suitable support to prevent foot drop and the nutrition of the muscles is maintained by massage and electrotherapy.
2. Nerve repair. Great care must be taken to suture the ends of the nerve accurately so that the lateral and medial popliteal segments may be properly opposed. A gap can usually be bridged by flexing the knee and extending the hip. After operation the knee is kept flexed for 3 weeks and is then gently and gradually extended.
3. Orthopaedic treatment. In cases of persistent paralysis great improvement in gait can be obtained by the use of a suitable hinged knee-splint and by arthrodesis of the ankle or tendon fixation. Amputation through the leg is indicated only for persistent severe pain or extensive trophic ulceration.

Lateral popliteal nerve

Aetiology

1. Deep wounds of the thigh.
2. Fractures of the upper end of the fibula.
3. Subcutaneous tenotomy of the biceps tendon.
4. Pressure by strapping, bandages, splints and plasters.

Clinical features

Motor

There is paralysis of the extensor and peroneal muscles. In the early stages a drop foot deformity develops but later on contraction of the opposing muscle groups causes paralytic talipes equinovarus.

Sensory

There is anaesthesia of the dorsum of the foot and the front and outer side of the lower two-thirds of the leg.

Examples of nerve injuries which may occur under anesthesia

- Faulty injection or extravasation of nerve irritants as thiopental or propofol.
- **Brachial plexus** injury when the arm is suspended at an angle more than 50 degrees to the operating table with external rotation causing overstretch of the roots. Allowing the arm to drop off the operating table, overextension of the arm with the head tilted to the opposite side or compression of the plexus by shoulder bracelets in the Trendelenburgh position produces similar overstretch.
- **Ulnar nerve** injury may occur when the arms are kept pronated by the side of the patient. In this position, the nerve may be compressed between the hard edge of the operating table and the middle epicondyle.
- **The radial nerve** may be compressed between the hard edge of the operating table and the humerus in the spiral groove. Tourniquet injury and faulty intramuscular injections in the arm are possible causes of injury.
- **The sciatic nerve** may be compressed in the lateral position of hip surgery or intramuscular injections.
- **The common peroneal** nerve may be compressed between the operating table and the fibula. It is a rather common type of injury.
- **The tibial nerve** may be compressed in the popliteal fossa by the knee supports in the lithotomy position.

Autonomic nervous system

Surgical anatomy

The parasympathetic and the sympathetic systems balance and antagonize each other.

Parasympathetic system

This has its origin in the brain and sacral region. It consists of 2 portions

1. **Cranial outflow.** The cranial parasympathetic fibres pass through the oculomotor nerve to the pupil, the facial nerve to the salivary glands and the vagus to the heart, lungs, alimentary canal, liver and pancreas. They stimulate the contraction of the pupil and the secretion of saliva and are secretory and motor to the alimentary canal and inhibitory to the heart.
2. **Sacral outflow.** The sacral parasympathetic fibres pass via the second, third and fourth sacral nerves, to form the pelvic splanchnic nerve which lies close to the presacral nerve and runs upwards to the inferior mesenteric artery at its origin from the aorta. This nerve is concerned with the emptying processes and so it supplies motor impulses to the rectum and bladder and causes penile erection.

Sympathetic system

This system is composed of preganglionic fibres, ganglionated trunks, visceral ganglia and postganglionic fibres.

1. **Preganglionic fibres** (white rami communicants) are the axons of the cells of the lateral horns of the grey matter of all the thoracic and upper 2 or 3 lumbar segments of the cord. They are medullated and pass from the anterior roots of the thoracic and upper lumbar nerves to join the sympathetic chains. Some preganglionic fibres pass directly to the medulla of the adrenal gland which represents the synapse and postganglionic fibre.

2. **Sympathetic chains.** These are a pair of ganglionated trunks that lie on the sides of the bodies of the vertebrae and extend from the base of the skull to the coccyx. Each chain is formed of ganglia and fibres. There are 3 cervical ganglia, 11 or 12 thoracic, 4 lumbar and 4 sacral and the cords join over the coccyx to form the ganglion impar. The inferior cervical and first thoracic ganglia are closely connected together to form the stellate ganglion which lies in a groove on the neck of the first rib.
3. **Visceral ganglia and plexuses** contain the cells whose axons pass direct to the abdominal viscera. Their preganglionic fibres pass, without synapsing, through the sympathetic chains. The main visceral ganglia are the coeliac, the superior mesenteric and the inferior mesenteric.
4. **Postganglionic fibres** are non-medullated fibres which arise from cells of the ganglionated trunk and visceral ganglia and pass to the skin vessels and viscera.

Sympathectomy

Indications

1. Vascular conditions. Sympathectomy interrupts the vasomotor fibres and causes dilatation of blood vessels and improvement of the local circulation. It is, therefore, useful in the following conditions
 - a. Raynaud's disease. Sympathectomy is very effective in the early stages. Pain is completely relieved but attacks of cyanosis may still occur.
 - b. Buerger's disease. The operation relieves the pain and promotes healing of ulcers but the prognosis is guarded since relapses after 2 or 3 years of improvement are common.
2. Hyperhidrosis. Excessive sweating of the hands is promptly relieved by cervicodorsal sympathectomy, whereas that of the feet responds to lumbar sympathectomy.

Principles of cervicodorsal sympathectomy

- The sympathetic supply of the upper limb is derived from the second and third thoracic nerves. The preganglionic fibres enter the corresponding ganglia and ascend in the sympathetic trunk to synapse in the first thoracic ganglion (a few synapse in the second ganglion from which the postganglionic fibres proceed in the nerve of Kuntz to the first thoracic nerve). To denervate the upper limb the second and third thoracic ganglia are excised.
- The sympathetic trunk below the third ganglion is divided together with the rami communicantes of the second and third ganglia and the nerve of Kuntz. The stellate ganglion is left intact to avoid Homer's syndrome.
- The operation can be performed through on the following approaches
- Anterior approach through a transverse incision in the lower part of the neck.
 - Axillary approach.
 - Thoracoscopy
 - The operation can be done laparoscopically.

Principles of lumbar sympathectomy

- The preganglionic fibres for the lower limb pass through the lower thoracic and upper two lumbar nerves to the corresponding ganglia of the sympathetic trunk and descend in the trunk to synapse in the lower lumbar and sacral ganglia.
- Excision of the lumbar chain constitutes a preganglionic denervation of the lower limb.
- To perform sympathectomy for the lower limb the upper 3 lumbar ganglia are excised. If bilateral sympathectomy is required, the first lumbar ganglion should be preserved on one side, otherwise failure of ejaculation occurs.

Nerve tumours

Neurofibromatosis

Neurofibromatosis is a proliferative condition of the endoneurium of nerves associated with tumour formation and sometimes with pigmentation of the skin. Several varieties of the disease are encountered either singly or in combination.

Solitary neurofibroma

This occurs most often between the ages of 20 and 50 years and usually affects the nerves of the upper limb but may occur in the spine, mediastinum or viscera. It forms a small elongated firm tender swelling very liable to cystic degeneration. The tumour should be completely excised.

Generalized neurofibromatosis (von Recklinghausen's disease)

Grossly

Generalized neurofibromatosis is a widespread affection of the cerebrospinal nerves as well as of the nerve roots within the cranial cavity and spinal canal and the nerve fibres in the skin, muscles and bones. The affected nerves are diffusely and irregularly thickened with the formation of tumour-like swellings.

Histologically

There is proliferation of the cells of the endoneurium. The tumour is composed of connective tissue fibres arranged in strands, bundles and whorls with little or no intercellular substance. The nuclei are elongated and hyperchromatic and are often arranged in rows to produce a palisade appearance. The nerve fibres may traverse the substance of the tumour or may be displaced to one side but they are not the seat of degeneration and so no sensory or motor changes are observed. Myxomatous degeneration is common and sarcomatous changes (secondary neurosarcoma) may follow, particularly after trauma or incomplete removal.

Clinical features

- **The disease** is often familial and usually begins in adolescence, but is commonly preceded with localized areas of pigmentation in the skin (café-au-lait patches).
- Multiple tumours appear all over the body surface (Fig. 18.17) and grow slowly to form fusiform swellings with the long diameter in the axis of the affected nerve. They vary greatly in size, the largest usually lying in relation to the plexuses at the roots of the limbs. They are firm or soft in consistency and movable in a lateral direction but not in the line of the nerve.
- The affected nerves may or may not be palpably thickened but pain is usually absent.
- When a spinal nerve root is affected it may grow into the spinal canal producing the signs of an intradural or extradural tumour or into the mediastinum with signs of



Fig. 18. 17. Generalized neurofibromatosis



Fig. 18. 18. Plexiform neurofi-

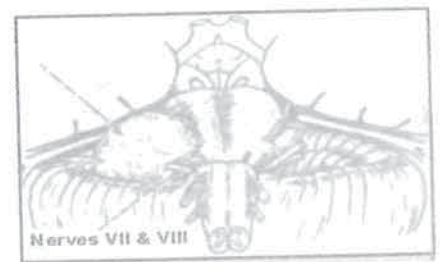


Fig. 18; 19. An acoustic neuroma is situated in the cerebellopontine angle. It affects functions of both 7th and 8th nerves

mediastinal tumour and sometimes it enlarges in both directions as a dumb-bell tumour.

- Malignant change is associated with pain, anaesthesia and paralysis in the territory of the affected nerve and progressive increase in size of the lesion.

Treatment

Excision of all tumours is impossible and the operation is indicated only for very large tumours, painful tumours and tumours producing pressure symptoms. The tumours should be completely resected.

Cutaneous neurofibromatosis (Molluscum fibrosum)

Multiple soft fibrous swellings arise in connection with the terminal filaments of the cutaneous nerves. They vary in size from a pin's head to a lemon size or larger and may be sessile or pedunculated. They are most numerous over the scalp, limbs, abdomen and back but the palms of the hands and the soles of the feet escape. In most cases, ie skin is the seat of pigmented areas (café-au-lait patches) or frank melanomata. Large and unsightly tumours are excised.

Plexiform neurofibroma

There is diffuse fibromatous thickening of the branches of a nerve forming a beaded swelling under the skin. The condition occurs most often in the subcutaneous tissues of the head and neck, the large nerves of the limbs and the autonomic plexuses of the abdomen. The overlying skin is often pigmented and thickened and may hang down in pendulous folds (pachydermatocele, Fig. 18.18). In more than half the cases manifestations of generalized neurofibromatosis are present.

Elephantiasis neuromatosa

This variety is related to and may co-exist with cutaneous neurofibromatosis. It appears in childhood and usually affects one of the extremities, especially the lower. The affected part gradually increases in size and may become enormous. The skin and subcutaneous tissues are greatly thickened. A variable number of tumours may be present and the skin may be pigmented and hairy.

Acoustic neuroma

In rare cases, neuro-fibromatosis may be associated with acoustic nerve tumours (Fig. 18.19). The disease has a striking familial incidence showing itself as a Mendelian dominant trait. It is often associated with tumours of the dura and choroid plexus.

Neurofibrosarcoma

This may arise de novo in an apparently normal nerve or may develop by malignant degeneration in a neurofibroma. The tumour is usually of a spindle-cell or myxosarcomatous character. It forms a painful rapidly growing tumour which invades the surrounding tissues, produces anaesthesia and paralysis and forms distant metastases. Wide excision should be carried out in localized tumours but amputation is indicated for extensive tumours and post-operative recurrences.

Ganglioneuroma

Pathology

Ganglioneuroma is a benign tumour which arises from the sympathetic trunks or adrenal medulla. It is composed of uni-or multipolar ganglion cells scattered among non-medullated nerve fibres. The tumour varies in size from that of a pea to that of a large melon, and is well-encapsulated.

Clinical features

The tumour occurs in childhood and forms a rounded or lobulated soft swelling which attracts attention by its size or pressure effects.

Treatment

The tumour is well-encapsulated and can be enucleated easily.

Neuroblastoma**Pathology**

Neuroblastoma is a highly malignant tumour which arises from the sympathetic chain or the adrenal medulla. It is composed of small round cells with hyperchromatic nuclei arranged diffusely or in rosettes. Ganglion cells and nerve fibrils are often present. The tumour forms a reddish purple soft swelling liable to haemorrhage and degeneration. It grows rapidly, reaches a large size and metastasizes widely by blood and lymph vessels.

Clinical features

The tumour usually occurs in early childhood and is most common in the abdomen and neck. It forms a large retroperitoneal mass and soon gives rise to metastases.

Treatment

The tumour is highly radiosensitive but metastases are so early and widespread that the prognosis is not favourable.

SCALP, SKULL AND BRAIN

Scalp

Anatomy of the scalp

The scalp is made of the soft structures that cover the skull from one temporal line to the other and from the eyebrows to the superior nuchal line. It is made of the following layers: (Fig. 19.1)

1. Skin.
2. Superficial fascia: This is a fibrous, tough layer of connective tissue containing the blood vessels and nerves of the skin. It is closely bound to the epicranial aponeurosis.
3. Occipito frontalis muscle: It has two pairs of bellies (frontal and occipital) united by the epicranial aponeurosis (galea aponeurotica) which is a long wide and thin tendinous sheet overlying the top of the skull.
4. Loose areolar tissue: This is a loose connective tissue layer with few blood vessels.
5. Pericranium: This is the periosteum on the outside of the skull.

CHAPTER CONTENTS

- Scalp
- Skull
- Intracranial injuries
- Hydrocephalus
- Brain abscess
- Intracranial tumours

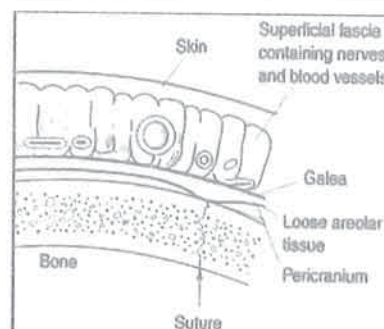


Fig. 19.1. Scalp layers.

Anatomical facts of surgical importance

1. The scalp is very richly supplied by blood vessels and so wounds of the scalp, even if lacerated, heal nicely and usually without infection.
2. The arteries present in the subcutaneous layer are adherent to the fibrous tissue of this layer. When a vessel is injured, the muscular coat of the divided artery cannot retract readily and so bleeding is very profuse. **How to stop bleeding from a scalp wound?**
 - a. Direct pressure. A depressed fracture should be excluded before applying direct pressure.
 - b. Applying multiple Allis forceps to the edge of the wound and bending them.
 - c. Applying multiple artery forceps to the galea aponeurotica and allowing them to fall back over the skin edge (Fig. 19.2).
 - d. All these measures are temporary before suture of the wound.
3. The skin, subcutaneous tissue and epicranial aponeurosis are firmly attached to each other, but they are loosely connected to the pericranium by a layer of loose areolar tissue. Complete avulsion of the scalp can occur.
4. The layer of loose areolar tissue allows accumulation of large amounts of blood or inflammatory exudate.

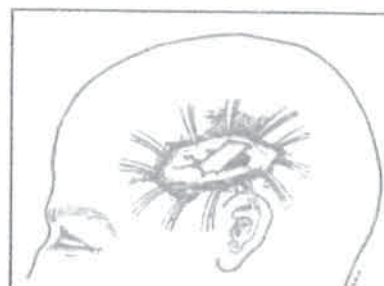


Fig. 19.2. Stopping scalp bleeding.

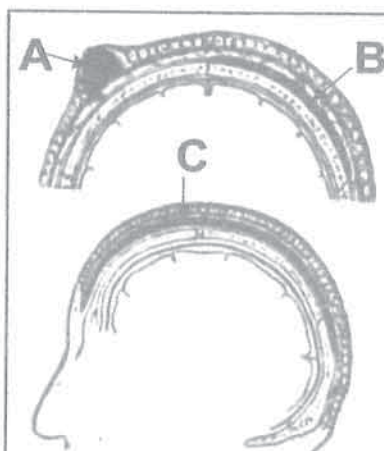


Fig. 19.3. Types of scalp haematoma.

Scalp injuries

Wounds of the scalp

These may be contusions, haematomas, lacerations or incised wounds.

- They are caused by sharp or blunt instruments or falls on the head.
- Bleeding is very excessive to the extent of causing shock.
- Healing is rapid.
- If infection occurs in the area of loose areolar tissue, extensive cellulitis is likely to occur.

Management

1. If there is a possibility of a skull fracture, plain skull X-ray is performed.
2. Closure is in 2 layers; galea to galea by absorbable sutures and skin to skin by non absorbable sutures. If there is liability to infection, closure is done in one layer of loose sutures. When there is a scalp defect, a rotational flap is performed.

Haematoma of the scalp

This may occur in the subcutaneous, subgaleal or subpericranial layers. Table 19.1 illustrates the differences between the 3 types (Fig. 19.3).

Treatment. Aspiration and a pressure bandage for subgaleal and subpericranial haematomas. A subcutaneous haematoma will resolve spontaneously.

Table 19.1. Types of skull haematomas

Subcutaneous haematoma	Subgaleal haematoma	Subpericranial haematoma (cephalhaematoma)
Confined to dense subcutaneous layer	Occupies loose areolar tissue under epicranial aponeurosis	Under the Periosteum
Due to direct blows		Injury to the head during delivery
Small, painful and moves with the scalp over the skull	Large, soft fluctuating swelling reaching anteriorly to the supraorbital ridges, posteriorly to the nuchal lines, and laterally to the temporal lines. The scalp floats over the swelling	Haematoma is limited by the suture lines to the underlying bone usually the parietal

Infections of the scalp

These are usually secondary to wounds. An abscess may occur in the subcutaneous, subgaleal or subperiosteal layers. Intracranial extension of infection from the subgaleal area may occur via the emissary veins. An infected sebaceous cyst is commonly seen in the scalp.

Treatment. Antibiotics according to culture studies. An abscess should be drained.

Tumours of the scalp

Benign	Lipoma-Papilloma-Plexiform	neurofibroma-haemangioma-Cirsoi
aneurysm.		
Locally malignant	Basal cell carcinoma.	
Malignant	Epithelioma-Melanoma-Fibrosarcoma-Sebaceous	adenocarcinoma
Metastases.		

Skull

Tumours of the skull

Benign:

These are rare. An ivory osteoma occasionally arises in the region of the frontal sinus.

Malignant:

These are either primary as, osteosarcoma, fibrosarcoma, giant cell tumour and multiple myeloma, or metastases from the breast, thyroid, adrenal, kidney and prostate.

Fractures of the skull

Fractures can occur in the vault or the base of the skull.

Fractures of the vault

Fractures of the vault may be depressed or fissure and any of them may be closed or open.

Aetiology

1. Local indentation of the skull by direct blows. This usually causes closed or compound depressed fractures.
2. General deformation of the skull due to compression by a hard flat surface. The skull yields at the point of maximum convexity, giving rise to a fissure (linear) fracture, often associated with brain injury.
3. Missile injuries.

Clinical features

Fractures of the vault may be fissure or depressed and any of them may be closed or open.

- **Fissure fracture.** The patient usually presents with the clinical picture of the associated cerebral damage.
- Plain x-ray (Fig. 19.4) will reveal the fracture, which may simulate a suture line of the skull but the latter exists at certain sites and it has a serrated edge.
- **Closed depressed fractures.** These are rare in adults, there is usually an overlying haematoma which may obscure the fracture. The depressed segment rarely causes cerebral compression. Plain x-ray will visualize the depressed segment.
- **Compound depressed fractures** (Fig. 19.5). There may be profuse bleeding, leakage of CSF and prolapse of a portion of the brain. Concussion is surprisingly slight and there is usually no compression. The main hazard here is the liability to infection.

Complications of a depressed fracture

1. Dural tear leading to prolapse of the brain.
2. Infection. This is very serious as it may lead to osteomyelitis or meningitis.



Fig. 19. 4; Fissure fracture.

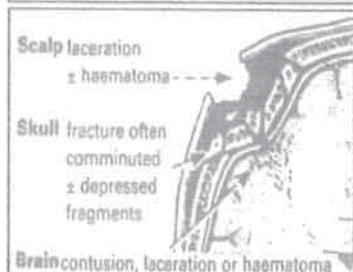


Fig. 19. 5. Compound depressed fracture.



Fig. 19. 6. Elevation of a depressed bone segment.

The most important cover to the brain is the dura and every attempt should be made to preserve and close it.

3. Epilepsy either early or late. Elevation of the depressed segment may diminish, but does not abolish this complication.
4. Cosmetic deformity.
5. Severe bleeding from one of the venous sinuses.

Treatment

Fissure fracture. Treatment of the associated cerebral damage follows the usual lines. The site of a fissure fracture, e.g. overlying the course of middle meningeal vessels should alert the surgeon to the possibility of an extradural haematoma. If the fracture is compound, the scalp wound is treated as usual.

Closed depressed fractures. These are treated essentially by conservative measures. Surgery to raise the depressed fracture is indicated in

1. Large depressed segment more than one inch with possibility of a dural tear.
2. If the depressed segment compresses an important area as the motor area or the speech centre.
3. If the depressed segment is causing a cosmetic deformity, e.g. in the frontal bone.
4. If the fracture is overlying an air sinus.

Compound depressed fractures These patients are operated upon in theatre under absolute aseptic precautions

1. Foreign bodies are meticulously removed.
2. The depressed segment is gently elevated to avoid tearing of the dura (Fig. 19.6).
3. Any prolapsed or necrotic brain tissue is sucked and haemostasis is performed.
4. Any dural tear is repaired.
5. Removed bone segments are cleansed and replaced.
6. The pericranium and the scalp are sutured.
7. Prophylactic antibiotics are administered.

Fractures of the base

Aetiology

Most fractures of the base are due to indirect violence caused by falls or blows on the vault producing general deformation of the skull. This leads to compression of the skull at one area, and giving way of the skull in the opposite. As the vault is elastic, it escapes while the base being rigid and weakened by multiple foramina, breaks. Sometimes, the fracture starts in the vault and extends to the base (Fig. 19.7).

Clinical features

The essential features are

1. Escape of cranial contents, e.g. blood, CSF or brain matter.

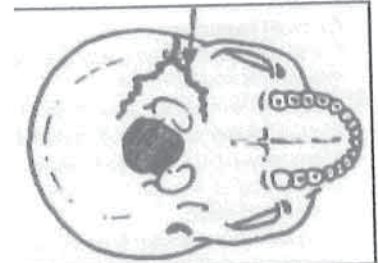


Fig. 19. 7. Extension of a vault

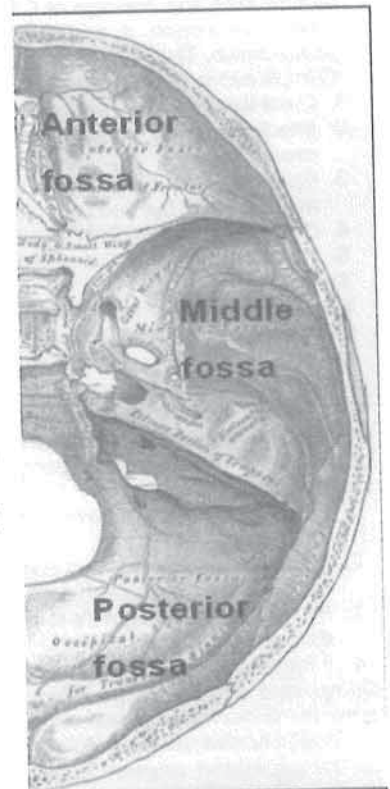


Fig. 19. 8. Cranial fossae

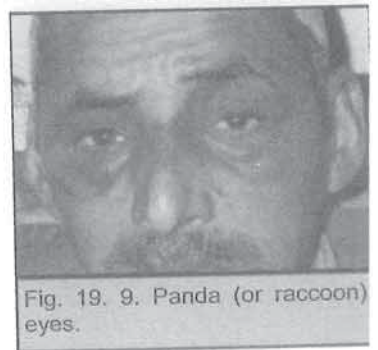


Fig. 19. 9. Panda (or raccoon) eyes.

2. Injury to the cranial nerves. All cranial nerves are liable to be injured except the twelfth, because the condyloid foramen is protected by a stout ridge of bone which deflects the line of fracture into the foramen magnum.
3. Signs of brain injury. The signs and symptoms in each cranial fossa (Fig. 19.8) are

Anterior fossa

1. Epistaxis occurs if the cribriform plate of the ethmoid is involved, or the blood may be swallowed to be vomited later on.
2. Extravasation of blood. If the fracture extends into the orbit, an effusion of blood follows, appearing first under the galea and later in the lids. "Panda bear" or "raccoon sign". This should be distinguished from an ordinary black eye by the following features:
 - a. There is no bruising of the skin around the orbit.
 - b. The effusion occurs several hours after the injury and commences in the lower lid before the upper.
 - c. The eye is sometimes pushed forwards, as the extravasation occurs into the tissues at the back of the orbit.
 - d. The extravasation may impede the action of the ocular muscles with limitation of movements of the eyeball.
 - e. If blood extends in subconjunctival space, its posterior limit cannot be seen. This sign differentiates it from subconjunctival haemorrhage due to direct trauma (Fig. 19.10).
3. Rhinorrhoea If the dura is torn, CSF trickles through the cribriform plate, and the patient may complain of a persistent salt taste due to the high chloride content of the fluid. The rhinorrhoea may persist for several weeks, and air may enter the cranial cavity when the patient blows his nose (pneumocephalus).
4. In severe injuries brain matter may escape into the nose or pharynx.
5. Injury to nerves The olfactory nerve is frequently torn, but unless its fellow is also damaged, the partial anosmia may pass unrecognized. The optic nerve usually escapes, but the third, fourth, sixth and first division of the fifth nerves may be injured at the sphenoidal fissure. Third nerve palsy produces a dilated pupil in a conscious patient.
6. Concussion is usually severe, but the intracranial damage associated with fractures of this fossa tends to be less severe than in the case of other fossae.

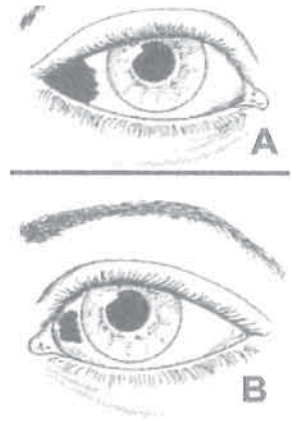


Fig. (19.10) Subconjunctival haemorrhage. A. Anterior cranial fossa fracture

Common features of skull base fractures

1. Escape of intracranial contents.
2. Cranial nerve injury.
3. Brain injury.

They are usually secondary to a major trauma and are, therefore, usually accompanied by a serious brain injury. They are often compound fractures and the risk of infection is high.

Middle fossa

1. Escape of CSF and blood from the ear is the most common and characteristic sign but should be differentiated from the bleeding of ruptured drum. Blood from torn tympanic membrane clots rapidly, but blood mixed with CSF continues to drip for several days without clotting.
2. Epistaxis occurs if the fracture involves the nasal sinuses, for the sphenoidal region is often involved and the cavernous sinus or internal carotid artery may be injured.

3. Surgical emphysema of the scalp may occur around and behind the ear if the fracture involves the mastoid antrum or air cells. Discoloration may appear over the mastoid process, Battle's sign (Fig. 19.11).
4. Injury to nerves. The facial nerve is commonly injured at the time of the accident, the nerve being torn with permanent paralysis. Occasionally, the paralysis appears after a few days from compression by extradural haemorrhage, and recovery often follows. If the paralysis develops some weeks after injury, it is produced by pressure from fibrous tissue or callus, and the condition will be permanent. The eighth nerve is sometimes involved, and occasionally the sixth nerve and the second and third divisions of the fifth.
5. Concussion is always present, often with signs of severe intracranial damage.

Posterior fossa

1. Extravasation of blood often occurs in the suboccipital region, producing a boggy swelling or discolouration at the nape of the neck. External haemorrhage does not occur because the fracture is rarely compound.
2. Injury to nerves. The ninth, tenth and eleventh nerves are occasionally damaged at the jugular foramen, but the hypoglossal nerve usually escapes.
3. The upper cervical nerves are sometimes irritated by blood producing retraction of the head and stiffness of the cervical muscles.
4. The patient is usually deeply unconscious as the pons and medulla may be injured, or subtentorial haemorrhage may give rise to severe bulbar compression, so that death frequently supervenes rapidly.

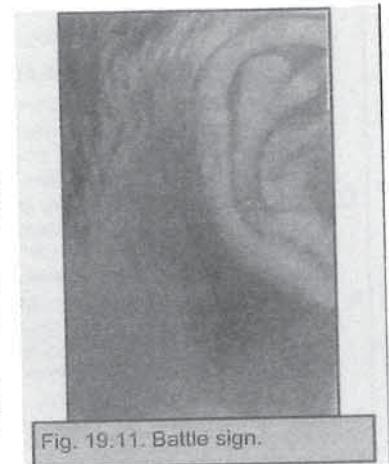


Fig. 19.11. Battle sign.

Treatment

1. Prevention of infection. Prophylactic antibiotics should be started at once and continued for one week after cessation of bleeding or CSF leakage. It is unwise to employ mouth washes or to syringe the ear, as this increases the risk of infection. When the ear is involved, it is gently sponged daily with cotton wool.
2. Control of CSF leakage, the patient should be propped up in bed to diminish the escape of CSF from the nose or ear. Usually the leakage stops in 85% of cases of rhinorrhoea and in 95% of cases of otorrhoea. If the discharge persists for more than 10 days, repair of the dura should be performed.
3. Treatment of the associated brain injury follows the usual lines.

Intra-cranial injuries

Cranio-cerebral injuries constitute one of the most commonly encountered injuries in casualty departments. With proper and diligent care many of these patients can be saved.

Aetiology

- Blunt injuries these may be of a low velocity as in falls or of a high velocity as in car accidents.
- Penetrating injuries as in missiles.

Mechanism of Injury

The main factor which leads to most of the sequelae of head injuries is the displacement and distortion of cerebral tissues occurring at the moment of impact.

Factors that affect severity of cerebral injury

1. Distortion of the brain. Posterior displacement of the cerebral hemisphere leads to distortion at the region of the hypothalamus and brain stem, while anterior displacement causes distortion of the corpus callosum. This distortion produces widespread damage to neurones, nerve fibres, glia and blood vessels. Distortion of the brain stem affects the reticular formation leading to temporary or persisting loss of consciousness.
2. Mobility of the brain in relation to the skull and membranes. Acceleration or deceleration injuries cause considerable movement between the brain and the dura leading to more brain damage.
3. Configuration of the interior of the skull. Smooth areas of the skull will cause less damage to the underlying brain than rough or sharp areas, e.g. the temporal lobe may be damaged by the sharp sphenoid ridge and the frontal pole by the rough floor of the anterior cranial fossa.
4. Age of the patient. A young patient will have a better chance of recovery than an elderly one as the functional reserve of the brain is higher.

Pathology

Pathologically the effects of head injuries can be classified into two groups

Primary pathological sequelae

Brain concussion, contusion and laceration represent different degrees of brain damage which occur at the moment of impact.

1. **Cerebral concussion.** There is slight brain distortion without any organic structural damage, leading to temporary loss of consciousness, followed by complete recovery. Immediately after the injury, the patient loses consciousness, the muscles are relaxed, the pulse is rapid and weak and the respiration is shallow and slow. The pupils may be contracted and reactive and the reflexes are absent or sluggish. In uncomplicated cases within minutes, rarely hours, complete and perfect recovery occurs.
2. **Cerebral contusion.** There are areas of bruising and swellings with intact pia arachnoid, localized or generalized oedema and haemorrhage due to tearing of blood vessels. Clinically there is a prolonged period of unconsciousness and there are physical signs of focal neurological damage. On recovery of consciousness, the patient complains of headache, photophobia, and confusion.
3. **Cerebral laceration.** The pia arachnoid is torn, with bloody effusion in the CSF.

Secondary pathological sequelae

1. **Brain oedema.** Intracellular and extracellular accumulation of fluid occurs. This oedema may be localized around the area of brain injury or it may extend to involve the whole brain leading to marked rise of intracranial pressure. Clinically this simulates the picture of brain compression due to a haematoma, yet without focal manifestations.
2. **Intracranial haemorrhage.** Arterial or venous bleeding occurs leading to an intracranial haematoma which may be extradural, subdural or intracerebral. Deep

intracerebral haematoma are usually secondary to a severe trauma causing tearing of many small vessels and are commonly associated with serious brain damage, oedema and necrosis.

3. **Vascular changes.** The rising intracranial pressure disturbs cerebral blood flow, with resultant ischaemic necrosis and subsequent brain oedema.

Cerebral infusion = [B.P — intracranial pressure].

4. **Herniation.** The rise of intracranial pressure will cause herniation of the contents of the supratentorial compartment through the tentorial hiatus, or contents of the infratentorial compartment through the foramen magnum

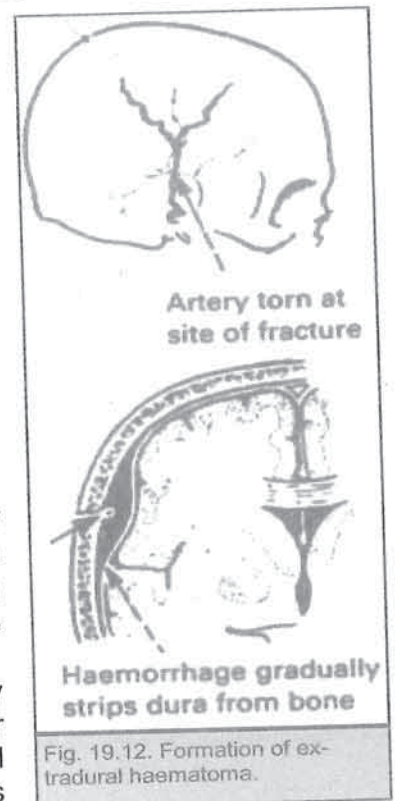
- a. **Tentorial herniation:** There is herniation of the medial part of the temporal lobe on the side of the supratentorial mass which causes compression of the oculomotor nerve (constriction and then dilatation of the ipsilateral pupil) and the midbrain. Compression of the descending motor pathways from the opposite hemisphere leads to hemiplegia on the same side of the haematoma.

- b. **Foramen magnum herniation:** Compression of the vital centres in the medulla leads to decrease of the pulse and respiratory rates and elevation of the blood pressure (Cushing triad). Severe headache and neck rigidity are present.

5. **Brain stem injury.** This term denotes either primary damage to the brain stem (medulla and pons) or secondary injury due to unrelieved supratentorial herniation. The patient is unconscious with spontaneous extension spasms of all four limbs, opisthotonos, tachycardia, small pupils, pyrexia and rapid shallow treatment, the patient may recover but there is often marked spasticity.

6. **Infection.** This complication is liable to occur in patients with compound fractures or fractures of the base especially if there is CSF rhinorrhoea or otorrhoea. The usual complication is meningitis which may develop 2-3 days after the injury and manifests by fever and neck stiffness. Meningitis can be confused with subarachnoid haemorrhage. Diagnostic lumbar puncture will establish the diagnosis. Brain abscess is a possible sequel of infection.

7. **CSF rhinorrhoea.** This occurs secondary to a fracture involving the paranasal sinuses, frontal, ethmoid or sphenoid associated with a dural tear. A piece of brain tissue is forced into the dural tear and prevents its healing. This complication is liable to be followed by meningitis. The patient is treated initially by antibiotics. Indications for surgical interference include persistence of the rhinorrhoea more than 10 days, the presence of a fracture involving the frontal or ethmoid sinus and the occurrence of meningitis.



breathing. With intensive

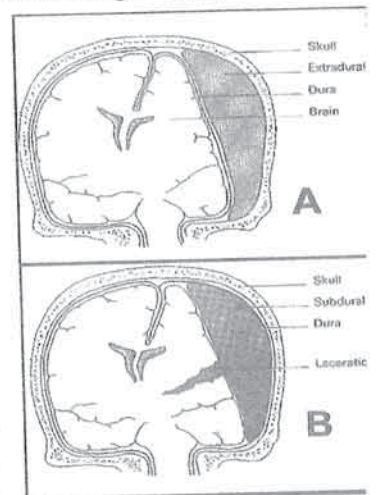


Fig. 19.13. A. Extradural haematoma B. Acute subdural haematoma

Extracranial factors that affect the cerebral injury

Certain extracranial factors may have a dramatic effect on the patient with a head injury

1. **Respiration.** Respiratory inadequacy causing hypoxia and increased PCO_2 can aggravate the development of severe brain oedema and venous congestion leading to irreversible changes. It is of utmost importance to keep the PCO_2 and PO_2 at normal levels in a patient with a head injury.
2. **Blood volume and blood pressure.** A fall in the cardiac output due to hypovolaemia may lead to irreversible cerebral ischaemic damage.
3. **Fluids.** Patients with cerebral injury have disturbances in the blood brain barrier and inappropriate secretion of ADH. Intravenous infusion of hypotonic fluids in these patients will lower the plasma osmolarity leading to increased brain oedema and swelling.
4. **Temperature.** A rise in body temperature will cause a further deterioration in neurological state due to increased metabolic demands and accumulation of metabolites.

Factors that raise the intracranial pressure in patients with head injury

1. Brain edema.
2. Intracranial haematoma extradural, subdural or intracerebral.
3. Cerebral swelling due to local vasodilatation and loss of autoregulation.
4. Rise of arterial PCO_2 .

Acute extradural haematoma

Aetiology

Usually this lesion is due to a trivial trauma applied to the side of the skull causing a fracture of the temporal or parietal bone. The dura is driven inwards and tears the anterior or posterior branches of the middle meningeal artery or vein leading to accumulation of blood in the extradural space (Fig. 19.12). Blood will pass in 3 directions

1. Outwards through the fracture to form a boggy swelling under the temporalis muscle.
2. Upwards over the parietal region.
3. Downwards into the middle fossa.

An extradural haematoma can occur without a skull fracture especially in children as they have an elastic skull.

Clinical picture

The clinical picture is usually described as passing into 3 stages.

1. **Stage of concussion.** The patient usually has a mild blow to the head (e.g., a ball hitting the skull) causing a brief period of concussion.
2. **Stage of lucid interval.** The patient recovers from the initial concussion and may even resume his activities. During this period blood is accumulating gradually in the extradural space.

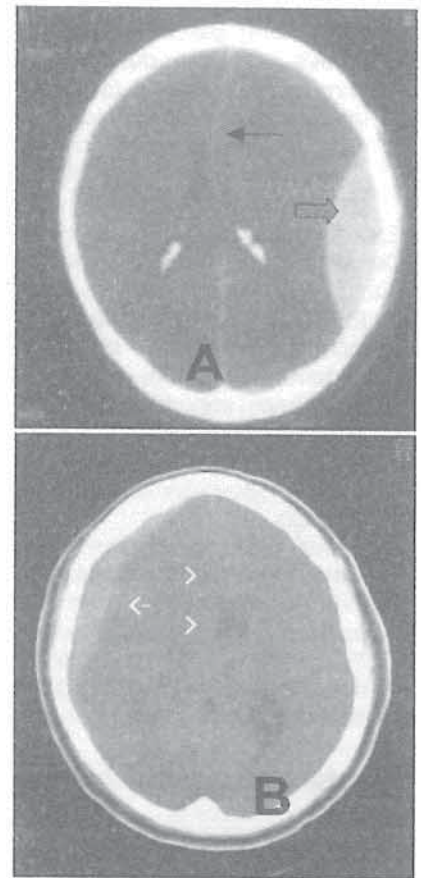


Fig. 19.14. On CT scan, acute haematomas appear hyperdense. There is usually shift of midline structures to the opposite side (arrows).
A. Extradural haematoma is bi-convex (lenticular).
B. Subdural haematoma is concavo-convex (crescentic).

3. Stage of compression.

- Gradual progressive deterioration in the level of consciousness is the salient feature. The patient becomes confused and irritable. Later he becomes drowsy, semicomatose or even comatose with stertorous breathing.
- Contralateral hemiparesis due to compression of the ipsilateral cerebral cortex.
- Tentorial herniation occurs. Compression of the oculomotor nerve causes constriction, rapidly followed by dilatation of the ipsilateral pupil. Later the contralateral pupil will constrict and then dilate.
- Continuous tentorial herniation forces the opposite crus against the rim of the tentorium, producing hemiparesis, which this time occurs on the same side as the haematoma.
- As the coma deepens the blood pressure rises and the pulse and respiration slow down. Finally hyperpyrexia, decerebrate rigidity and bilateral dilated fixed pupils occur, and constitute clinical evidence of bad prognosis.

It should be stressed that this classic picture is only found in a minority of patients. If the source of bleeding is venous, the lucid interval will be longer.

Acute subdural haematoma

This usually follows a severe blow to the skull leading to rupture of a large cortical vein as it crosses the subdural space to reach the fixed subdural venous sinuses (Fig. 19.15). The haematoma is less commonly due to a cortical laceration with subsequent haemorrhage. Clinically the patient may present with a picture similar to that of an extradural haematoma but certain differences exist (Table 19.2).

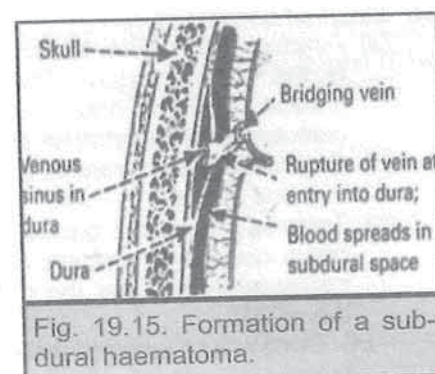


Fig. 19.15. Formation of a subdural haematoma.

Acute intracerebral haematoma

Table 19.2. Differences between extradural and acute subdural haematomas (Fig. 19.13 and 19.14)

Extradural haematoma	Acute subdural haematoma
Usually mild trauma	Severe trauma
Usually mild brain damage	Severe brain damage and laceration
Lucid interval may be present	Persistent loss of consciousness with no lucid interval
Usually unilateral	Commonly bilateral and extensive
Early surgery is successful	The patient has serious brain damage and oedema in addition to the haematoma, and so the results of surgery are not very successful. Mortality rate is up to 50%

This is the least common of traumatic intracranial haematomas. It is usually accompanied by brain contusions, laceration, oedema and necrosis and all these factors contribute to the poor general condition of the patient. CT scan should be performed. Whether to operate or not depends on the judgement of the surgeon but the results are unpredictable.

Management of intracranial injuries

Certain points have to be stressed.

1. The majority of patients with cerebral injury will need only conservative measures and not surgical interference. Unnecessary surgery may even worsen the condition of the patient.
2. The care of the patient, with a head injury is mainly the responsibility of the surgeon in the casualty department who may ask for a consultation of a neurosurgeon.
3. Many extracranial factors (blood volume, PO_2 , PCO_2 and the temperature) have a serious impact on the injured brain.

The management of patients with a head injury must follow a planned programme as follows

1. Dealing with the life saving priorities.
2. Initial examination.
3. Performance of the necessary investigations.
4. Continuing care and observations.
5. Possible need for surgery to evacuate an intracranial haematoma.

1. Dealing with life saving priorities

As in any injured patient, protection of the airway, maintenance of adequate breathing, arrest of haemorrhage and correction of shock have the first priority. It is to be stressed that patients with a head injury are more likely to die from airway obstruction than from any remediable intracranial lesions. The presence of shock in a patient with a head injury is most likely due to internal haemorrhage in the thorax or abdomen.

2. Initial examination

This is of utmost importance as it provides a base level for subsequent observations.

The patient is examined thoroughly as a whole, as in many cases there is an associated abdominal or thoracic injury. What to examine?

- (a) **Vital signs** pulse, B.P., and respiratory rate.
- (b) **Scalp** Far any scalp wound or haematoma.
- (c) **Skull** for any fracture.

- (d) **Assessment of level of consciousness:** A very useful and practical method is to follow "The Glasgow coma scale" (Table 19.3). The total points are added. The higher the score, the better is the prognosis. According to the scale cases of head injury are classified into mild (13-15), moderate (9-12) or severe with a score of 8 or less.

- (e) **Pupils:** Both pupils should be examined for their size and reaction to light. A pupil which is dilated immediately after the accident is most likely to be due to direct injury of the orbit or the oculomotor nerve, while a pupil which was constricted on initial examination and later dilates, denotes lateralization due to a supratentorial haematoma.

- (f) **Limbs:** Examine for any fracture or vascular injury. Hemiplegia in the acute phase is more likely to be due to primary cerebral damage rather than due to a compressing intracranial haematoma.

- (g) **Chest:** Examine for fractured ribs, pneumothorax, or haemothorax.

Table 19.3. Glasgow Coma Scale (GCS)

Eye opening	
▪ Spontaneous	4
▪ To verbal command	3
▪ To pain	2
▪ None	1
Best verbal response	
▪ Oriented	5
▪ Confused	4
▪ Inappropriate words	3
▪ Incomprehensible sounds	2
▪ None	1
Best motor response	
▪ Obeys command	6
▪ Localizes pain	5
▪ Flexion withdrawal	4
▪ Abnormal flexion (decorticate rigidity)	3
▪ Abnormal extension (decerebrate rigidity)	2
▪ None	1

- (h) **Abdomen** Examine for internal haemorrhage or peritonitis.
- (i) **Back** For the possibility of fractures or dislocations.

3. Indications for admission to the hospital of patients with head injury include

1. Any depression of level of consciousness.
2. Skull fracture.
3. Focal neurological signs.
4. Persistent headache or vomiting.
5. Absence of responsible relatives who can observe the patient for the first 24 hours.
6. Difficulty in assessing the patient; alcoholic, young or epilepsy.

The level of consciousness is the most important sign of patient's progress.

CT scan should be performed before surgery to evaluate the size of the haematoma and the degree of brain damage and oedema.

4. Investigations

After the patient is resuscitated and a patent airway is maintained, the surgeon should order the necessary investigations.

- (a) **Plain skull x-ray** Plain radiography can demonstrate the site and type of a skull fracture. This may give a clue to the severity of the case and to the possible site of an extradural haematoma which may develop later on. A foreign body can also be visualized.
- (b) **CT scan** is highly recommended for
 - a. Depressed or compound fractures.
 - b. Impaired level of consciousness or focal neurological signs.
 - c. Basal skull fractures.
 - d. Deteriorating level of consciousness.

CT scan will detect brain edema, brain contusion or laceration. The site, size and progress of the an intracranial hematoma can be assessed. Acute extradural or subdural hematomas are hyperdense compared to the brain, and there will be shift of mid line structures.

N.B. If the patient, on clinical grounds, is in urgent need for evacuation of an intracranial haematoma, no time should be lost in doing investigations, and surgery should be done immediately.



CT scan showing a frontal contusion

5. Continuing care and observations

The aim of conservative treatment is to give the patient the maximum care until spontaneous recovery occurs and to detect, at the earliest possible moment, the development of complications that may need surgical interference.

- (a) Attention to the airway as described before. If spontaneous respiration is inadequate to keep the normal levels of PO_2 and PCO_2 , an endotracheal tube is inserted and controlled ventilation started. An endotracheal tube needs full sedation or even muscle relaxants. It can be left for a period up to 7 days. If ventilation is needed for a longer period, a tracheostomy is performed.
- (b) A Foley catheter is inserted to facilitate the nursing care and to estimate the urine output.
- (c) Frequent change of posture to avoid bed sores.
- (d) Physiotherapy to the joints and massage to the muscles.

- (e) A nasogastric tube is inserted for feeding.
- (f) Osmotic diuretics: These raise the osmolarity of the plasma and so reduce the brain oedema. 250 ml of 20% Mannitol are given over a period of 20 minutes and may be repeated every 8 hours. Before mannitol is administered it is essential to exclude an intracranial haematoma and to check that the renal function is satisfactory. Mannitol should not be used for more than 48 hours to prevent rebound oedema and electrolyte disturbances.
- (g) Frusemide; 40-80 mg IM is an alternative to mannitol.
- (h) Corticosteroids. These are empirically prescribed but there is no clear evidence that they reduce the cerebral oedema nor, improve the outcome in patients with severe head injury.

6. Repeated observations for of the following

- Level of consciousness using the Glasgow coma scale.
- Pulse, B.P, and temperature.
- Respiration.
- Pupils.
- Reflexes.

Causes of deterioration of the patient

1. Brain oedema leading to increased intracranial tension
2. Airway obstruction and/or hypoventilation leading to brain swelling and increased intracranial tension. Respiratory insufficiency can be confirmed by estimating PO_2 and PCO_2 .
3. Intracranial haematoma. This can be confirmed by CT scan.
4. Fever due to respiratory infection or meningitis.
5. Overtransfusion by hypotonic fluids, or dehydration.
6. Epilepsy. If not accompanied by convulsions, it is difficult to differentiate epilepsy from an intracranial haematoma.

7. Surgery to evacuate an acute intracranial haematoma

Once the clinical picture of the patient deteriorates, the possibility of an intracranial haematoma is raised and it is of utmost importance to exclude or verify this possibility because surgical interference at this stage can save the patient, otherwise progressive deterioration and fatal herniation of the cerebellar tonsils and medulla through the foramen magnum will occur. A rising intracranial tension produces the following manifestations

1. Deterioration in the level of consciousness.
2. Slowing pulse rate.
3. Rising blood pressure.
4. Slowing respiration.

A high intracranial pressure is caused either by a haematoma or by cerebral oedema. CT scanning can easily diagnose the presence and site of an intracranial haematoma.

Operation

1. The operation is done under general anaesthesia with endotracheal intubation.
2. The patient is placed supine with the site of the haematoma uppermost, and the head is raised slightly to reduce venous bleeding.
3. A formal craniotomy by an osteoplastic flap is done (Fig. 19.16). The flap includes skin, temporalis muscle and part of skull. Four or five burr holes are done and are connected by a saw.

4. Skin and temporalis are not dissected from bone. The whole osteoplastic flap is turned down, thus exposing the dura.
 5. If an extradural haematoma is present, it can be removed by suction and the middle meningeal artery coagulated or under-run by a stitch.
 6. Occasionally it may be necessary to follow the middle meningeal artery to the foramen spinosum which is packed with bone wax.
 7. If a subdural haematoma is found, the dura mater is opened in a cruciate fashion to allow rapid decompression of the brain.
 8. A drain is placed and the flap is returned to its place and fixed by suturing temporalis fascia and skin.
- Prophylactic anticonvulsant drugs are prescribed for 6 weeks to guard against epileptic fits.



Fig. 19.16. Craniotomy.

Late complications of head injuries

1. Chronic subdural haematoma

This complication is especially liable to occur in elderly persons and alcoholics secondary to a slight blow to the head which may pass unnoticed. The sudden displacement of the brain causes rupture usually of the superior cerebral veins as they pass to the venous sinuses. Cerebral atrophy in elderly persons and alcoholics makes displacement of the brain easier. The result will be a collection of blood in the subdural space. This haematoma is bilateral in 50% of cases (Fig. 19.17).

Clinical picture

The interval between the trauma and the symptoms varies between weeks to months. The symptoms are vague and consist of chronic headache, mental apathy, slowing of cerebration and the patient may even develop stupor.

Physical signs may be absent or at most there may be unilateral or bilateral extensor planter response. Pupillary changes are late and denote impending conization.

The symptoms and signs wax and wane. The condition may be diagnosed as psychosis or cerebrovascular accident.

Investigations

- Fundus examination. Papilloedema is exceptional.
- CT scan. This will reveal the haematoma which is hypodense compared to the brain.

Treatment is by evacuation through burr holes.

2. Post-traumatic headache

Patients with serious head injury usually have some residual symptoms a headache, giddiness, impaired sleep and defective concentration. These patients need prolonged convalescence.

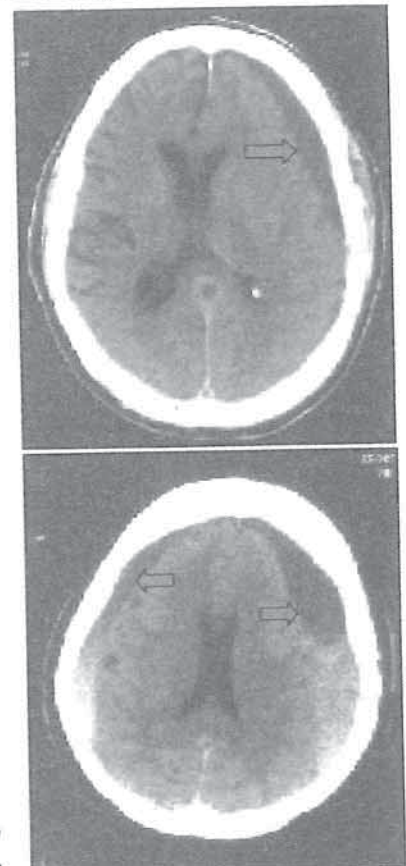


Fig. 19.17. CT scan showing chronic subdural haematomas. A chronic haematoma is hypodense. Upper scan. Unilateral haematoma with shift of midline structures to opposite side. Lower scan. Chronic bilateral subdural haematomas.

3. Post-traumatic epilepsy

This may be either

- (a) Early within one week after the injury. Its incidence is increased in patients with depressed fracture or intracranial haematoma. These patients need prophylactic anticonvulsants for 6 weeks.
- (b) Late epilepsy, the incidence of which is much higher in those who had early epilepsy. It occurs within 1-4 years after the injury. Treatment is by long term anticonvulsant drugs.

4. Post-traumatic hydrocephalus.

This may need shunt operation.

Hydrocephalus

Hydrocephalus is an abnormal accumulation of excess cerebrospinal fluid (CSF) within the ventricles and/or the subarachnoid space. It literally means 'water head'.

Normal CSF circulation

CSF is mostly produced by the choroid plexus in the lateral ventricles of the brain. From there it passes through the foramen of Monro on each side to reach the midline third ventricle. The fluid then passes downwards to the aqueduct of Sylvius to the fourth ventricle. The fourth ventricle has two lateral foramina (of Luschka), and one midline foramen (of Magendi), which transmit CSF to the subarachnoid space around the brain and spinal cord. Most of the fluid absorption occurs inside the skull through the arachnoid villi to the blood stream.

Because of the difference in aetiology, symptoms, and management; hydrocephalus is discussed under two major entities. These are hydrocephalus in children, and hydrocephalus in adults.

Hydrocephalus in children

Prevalence

Hydrocephalus occurs as an isolated congenital disorder in approximately 1/1000 live births. It also occurs in association with spina bifida in 1/1000 births in the United States.

Pathophysiology

The abnormally excessive CSF accumulation is typically associated with dilatation of the ventricles and increased intracranial pressure. Hydrocephalus usually results from an obstruction in the CSF pathway, and only rarely, from overproduction of CSF



Fig. 19.18. Hydrocephalus and the "setting sun" sign.

Classification

1. Non-communicating hydrocephalus results from lesions that obstruct the ventricular system either at the aqueduct or basal foramina.
2. Communicating hydrocephalus results from lesions that obstruct the subarachnoid space.

Aetiology

1. Aqueduct occlusion. This is the result of true aqueduct stenosis, subependymal gliosis (e.g., after toxoplasmosis), or aqueductal forking (a congenital anomaly).
2. Arnold Chiari malformation. Hydrocephalus is associated with myelomeningocele. The fourth ventricle is displaced caudally below the foramen magnum causing impedance to CSF flow to the subarachnoid space.
3. Dandy-Walker syndrome. A cystic dilatation of the fourth ventricle obstructs CSF flow.
4. Cerebral malformations as encephalocele, or hydrancephaly. Arachnoid cysts that obstruct the CSF flow.
5. Neoplasms that are mainly situated in the fourth ventricle (medulloblastoma, astrocytoma, and ependymoma).

Clinical features

- If hydrocephalus occurs before closure of the skull sutures, the infant presents with macrocephaly, tense nonpulsatile anterior fontanelle, distended scalp veins, and separated cranial sutures. The eyes show the sun set appearance with failure of upward gaze (Fig. 19.18).
- If the sutures have already closed, the child usually presents with headache awaking him from sleep, vomiting, drowsiness, blurred vision, and squint.

Investigations

1. X-ray of the skull demonstrates separation of sutures in infants, and the 'beaten silver' appearance in older children.
2. Cranial ultrasonography can be done through the anterior fontanelle and is useful for follow up.
3. CT and MRI are the investigations of choice in diagnosing hydrocephalus, as well as the site of obstruction and the causative pathology.

Treatment

No medication can treat hydrocephalus effectively.

The ideal management of hydrocephalus is the removal of the cause, but unfortunately, this is impossible in most cases of congenital hydrocephalus.

The current management of the problem is by CSF diversion using shunts. The most often used is the ventriculo-peritoneal shunt (Fig. 19.19) which consists of a ventricular catheter, a valve permitting CSF flow in one direction, and a distal peritoneal tube. The valve opens when the intracranial pressure exceeds a certain level, the excess fluid enters the peritoneum and is absorbed there to reach the systemic circulation. The ventricular catheter is inserted through a burr hole in the skull to reach the frontal horn of the lateral ventricle. The peritoneal tube is inserted in the epigastric midline below the xiphoid process.

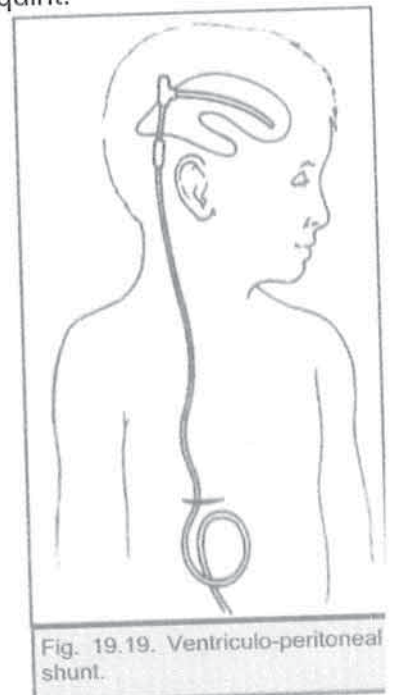


Fig. 19.19. Ventriculo-peritoneal shunt.

The commonest two complications of shunts are infection and obstruction. In case of shunt infection the shunt is removed, an external ventricular drain is applied, intravenous antibiotics are given for two weeks. After the infection subsides another shunt is inserted, usually on the other side of the head. In case of shunt obstruction the child develops increased intracranial pressure. The condition is managed by revising the shunt and replacing the malfunctioning component.

Hydrocephalus in adults

Classification and aetiology

Hydrocephalus in adults can be classified as acute or chronic, communicating or non-communicating, and normal pressure or high pressure. The common causes include

- (a) Non-communicating
 - a. Tumours or cysts of the ventricles.
 - b. Aqueduct stenosis.
 - c. Posterior fossa malformations and tumours.
- (b) Communicating
 - a. Trauma.
 - b. Subarachnoid haemorrhage.
 - c. Infection.
 - d. Idiopathic.
 - e. Extra-axial tumours.

Clinical features

- In acute hydrocephalus the patient presents with headache, vomiting, papilloedema, and drowsiness.
- In chronic and normal pressure hydrocephalus the patient presents with gait disorders, memory loss, urinary incontinence, and slowing of thought and action.

Investigations

- CT and MRI are the investigations of choice to diagnose hydrocephalus and its underlying cause.
- In case of normal pressure hydrocephalus an overnight monitoring of CSF pressure and perfusion is helpful to record bouts of raised intracranial tension. Access is gained through a ventricular or a lumbar catheter.

Treatment

As for children CSF diversion is done mainly by the ventriculo-peritoneal shunt and less commonly by the ventriculo-atrial or ventriculo-pleural shunts. Lumbo-peritoneal shunt can be done for the communicating variety of hydrocephalus.

Brain abscess

Despite advances in diagnosis and therapy, the incidence of brain abscess does not appear to be diminishing, and may, in fact, be increasing.

Aetiology and pathogenesis

Brain abscess can form through one of the following pathways:

1. The majority of brain abscesses arise by direct spread from paranasal sinuses, middle ear (Fig. 19.20), or mastoid infection.
2. Skull defects which are acquired (e.g. cholesteatoma of the middle ear), or congenital (e.g., dehiscence of the tegmen tympani).
3. Haematogenous dissemination from a primary site (e.g., from dental or tonsillar abscess) usually causes multiple brain abscesses (Fig. 19.21). This route is commoner in patients having congenital heart disease with right to left shunt.

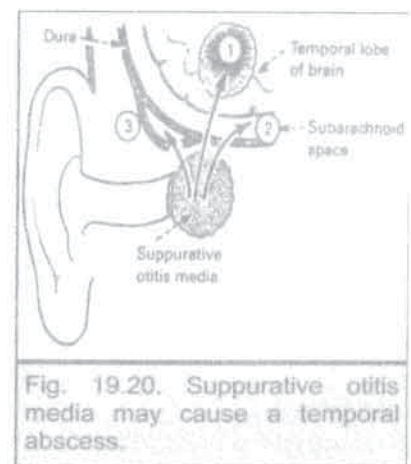


Fig. 19.20. Suppurative otitis media may cause a temporal abscess.

4. Post-traumatic (Fig. 19.22). The presence of foreign bodies increases the likelihood of brain abscess formation.
5. Previous craniectomy Immunocompromised patients (e.g., immunosuppressive therapy for transplantation, are more prone to affection.

Pathology

The formation of a collagen capsule in a developing abscess is the single most important response that limits the spread of infection to the rest of the brain.

Clinical features

No specific set of symptoms or signs is pathognomonic of brain abscess, which may present as a space occupying lesion without signs of systemic infection. The commonest symptom is progressively severe headache that is resistant to analgesics.

Fever is present in half of the cases and is usually of low grade. Other presentations include alteration of the conscious level in about 50% of the cases, and focal neurologic deficits.

Investigations

Laboratory findings

- Both the white blood cell count and the sedimentation rate are usually elevated.
- Lumbar puncture is contraindicated in case of a brain abscess to avoid a fatal conization.

Radiological

CT or MRI is the investigation of choice. The CT scan is performed with and without contrast. The MRI is done with gadolinium enhancement. They show a single (or multiple) space occupying lesion that is well delineated with an enhancing wall. The differential diagnosis of a single brain abscess in the CT and MRI is a solitary metastasis or cerebral infarction. In case of multiple abscesses the disease should be differentiated from multiple metastases and from tuberculomata.

Treatment

Non-surgical treatment

This is indicated for an abscess that is less than 1.5 cm.

1. Antibiotics: Appropriate antibiotic selection is based on culture and sensitivity results. However, while waiting for these results broad spectrum, high dose antibiotics are initially administered. The chosen drugs should have the ability to cross the blood-brain barrier. The usual regimen is a combination of penicillin G, trimethoprim-sulphamethoxazole, and an aminoglycoside. The majority of the responsible organisms are obligate anaerobes with 50% of them resistant to penicillin.
2. Corticosteroids: These are in common use to help reduce cerebral oedema, despite their potential adverse effect on the infection.

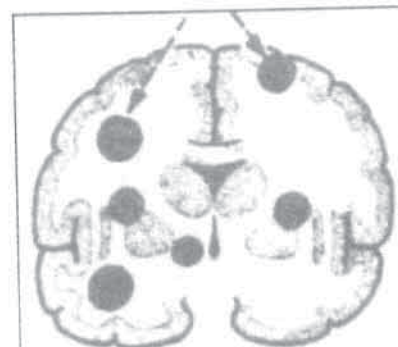


Fig. 19.21. Haematogenous abscesses are usually multiple.

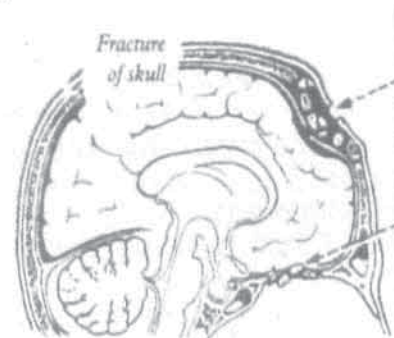


Fig. 19.22. Compound skull fractures, either of the vault or base, may produce a brain abscess.

Surgical treatment

Aspiration versus excision. The mortality rate resulting from aspiration alone, aspiration followed by excision, and primary excision alone, are almost similar. However, certain factors favour aspiration while others favour excision.

- Factors that favour aspiration

- Multiple abscesses.
- A deeply seated abscess.
- A critical location (e.g., motor or speech area).
- Poor general condition of the patient.

Aspiration is done by using a blunt-tipped needle by way of a burr hole in the skull.

- Factors that favour excision

- Multilocular abscess, as it is sometimes difficult to aspirate all the locules.
- A superficial abscess.
- The presence of a foreign body.
- Fungal abscesses, as they are known to have poor response to antifungal therapy.

Intracranial tumours

General considerations

The term intracranial tumour is used to refer to all neoplasms arising from the skull, meninges. Blood vessels, pituitary and pineal glands, cranial nerves, brain tissue, or congenital rests, as well as metastatic tumours.

Epidemiology

The commonest intracranial neoplasm is metastasis. In more than 20% of all patients with cancer, the brain and its coverings are involved by metastases at sometime in the course of the illness. In children, brain tumours are the second in frequency to leukaemia representing 22% of all childhood cancers.

Clinical features

1. May be symptomless.
2. General impairment of the cerebral functions such as amnesia, irritability, confusion, or dementia.
3. Headaches are present in about one third of tumour patients and are variable in nature and distribution. They may be related to increased intracranial pressure, or to direct stretch of the meninges.
4. Evidence of increased intracranial pressure as headache, vomiting, and blurring of vision. Usually the vomiting is not preceded by nausea, is projectile, not related to meals, and occurs in the early morning. Blurring of vision is related to increased pressure resulting in papilloedema and later optic atrophy.
5. Specific intracranial tumour syndromes are related to the site and nature of the tumour
 - a. Hemiplegia in tumours of the motor area.
 - b. Aphasia in tumours of the speech area.
 - c. Hormonal disturbances in functioning pituitary tumours (Cushing's disease, acromegaly, and galactorrhoea with prolactinomas).

Table 19.4. Classification of intracranial tumours

Secondary tumours	Commonly from bronchial and breast cancers
Primary tumours Glia tissue	Gliomas Astrocytoma Oligodendroglioma Ependymoma Glioblastoma multiforme
Meninges	Meningioma
Nerves	Neuromas, e.g., acoustic neuroma
Blood vessels	Haemangioblastoma
Embryonic	Medulloblastoma
Anterior pituitary	Pituitary adenoma Non-functioning adenoma Functioning adenoma
Maldevelopment	Craniopharyngioma (from Rathk's pouch)

Investigations

1. **Plain skull x-ray** Skull X-ray may be normal in the presence of a brain tumour, or it may show changes due to increased intracranial pressure in the form of
 - a. Prominent convolutional markings.
 - b. Thinning and erosion of the dorsum sellae.
 - c. Widening of the cranial sutures (in infants before their closure).
 Local changes in the plain skull X-ray may occur with specific tumours, e.g.
 - Supracellar calcification in craniopharyngiomas.
 - Hyperostosis and skull thickening in meningiomas.
 - Widening of the skull base foramina with skull base tumours.
2. **Computed tomography (CT).** Current equipment is capable of high spatial and contrast resolution. Intravenous iodinated contrast material is required in 60 to 80% of the examinations
 - Computed tomography accurately detects the site of the tumour.
 - It clearly demonstrates its boundaries.
 - It shows whether there is surrounding oedema or not.
 - It demonstrates any shift of midline structures by the mass.
 - It shows any associated hydrocephalus.
 - It shows any bony changes.
 - It gives a good idea about the nature of the tumour (solid, cystic, necrotic, or calcified), and consequently its pathology.
3. **Magnetic resonance imaging (MRI).** The idea is based on the fact that certain atoms with an odd number of nucleons inside their nuclei (e.g., H^1 , C^{13} , Na^{23}) behave like small magnets when placed in a strong magnetic field and line up in the direction of the field. The hydrogen nucleus with its single proton is the most typical of the body's elements to behave in this manner, and is thus the focus of current MR technology. Application of a radiofrequency pulse (RF) introduces energy into the sample of protons and displaces the net magnetic vector by an amount that is determined by the duration and amplitude of RF pulse. After the pulse is removed, the protons return to their original orientation and emit the absorbed energy in the form of an RF signal. The chemical state of the hydrogen atoms, and, thus, the surrounding tissue dictates the time it takes for the protons to "relax" within the magnetic field. The

RF signal is used by a computer to generate the MR image. There are no biological risks from magnetic resonance devices but MRI is contraindicated in the presence of metal implants as cardiac pacemakers, aneurysm clips, metallic foreign bodies and patient support systems as respirators or intravenous line stands. The material may move or stop working, thus endangering the patient's life. Material made of titanium permit safe MRI examination.

4. **Carotid angiography.** This is currently performed to evaluate a specific entity, usually of a vascular nature, or to supplement data provided by CT or MRI. It is usually performed by transfemoral catheterization. Angiography is an invasive technique that is used less frequently in modern neurosurgical practice.

Management

The following points are to be considered when dealing with an intracranial tumour.

1. If the patient shows signs of compression, urgent dehydration is started using diuretics as 25% dextrose, or mannitol. Dexamethazone is also very useful in the treatment of oedema surrounding brain tumours as it might restore a comatosed patient to consciousness while preparing for surgery.
2. If an intracranial tumour is associated with hydrocephalus, a finding that is common with posterior fossa tumours, the hydrocephalus is dealt with first by a ventriculoperitoneal shunt before surgery, or if needed after surgery, depending on the patient's general condition and the surgeon's experience.
3. Many of the skull base tumours that were considered difficult to reach are now easily accessible by removing as much as possible of the bones of the skull base, face, and ear in order to minimize brain retraction.
4. CT or MRI guided stereotactic surgery is done for biopsy or excision if the tumour is deeply seated in the brain. A head frame is used to facilitate very accurate three dimensional localization of the lesion, which can then be reached by fine instruments introduced through a burr hole.
5. In cases of malignant brain tumours or metastases the role of adjunctive therapy is of utmost importance to improve the prognosis. Radio and/or chemotherapy are supplementary to surgical excision.

The most significant findings pointing to an intracranial tumour, on physical examination, are the presence of papilloedema and signs of focal damage to the nervous system.
If a cerebral tumour is suspected lumbar puncture is contraindicated as it may precipitate a fatal conization of the brain stem through the foremen magnum.

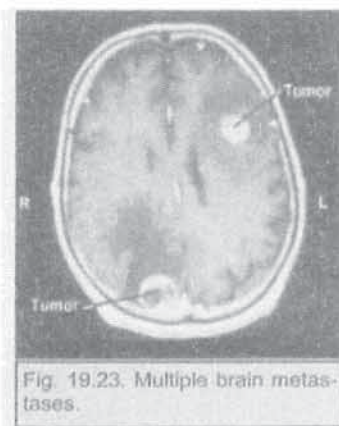


Fig. 19.23. Multiple brain metastases.

Individual intracranial tumours

Metastatic tumours

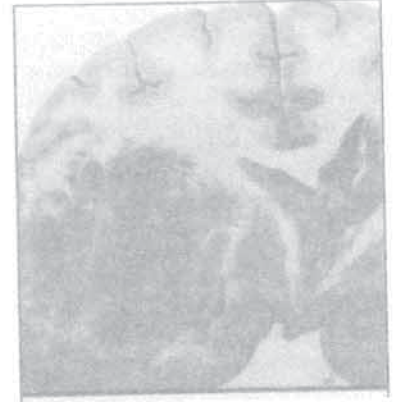
The following therapeutic approach to brain metastases is advocated

1. Multiple brain metastases (Fig. 19.23) are treated by brain irradiation.
2. Solitary brain metastasis
 - a. Disseminated disease or life expectancy less than 2 months Brain radiotherapy.

- b. Limited systemic disease and life expectancy more than 2 months
 - i. If the lesion is surgically accessible the best management is surgical excision followed by radiotherapy.
 - ii. If the lesion is inaccessible radiotherapy is the only treatment.

Gliomas

- Gliomas are central nervous system tumours that are derived from cells of glial origin. They are the commonest of primary CNS neoplasms.
- Tumours that are termed gliomas include the astrocytic tumours, oligodendrogliomas, ependymomas, and glioblastoma multiforme (Fig. 19.24).
- A biopsy or surgical resection is indicated in most cases to obtain a definite diagnosis. Except for low grade astrocytomas; gliomas cannot be cured by surgery alone. Adjuvant radio or chemotherapy is usually needed after biopsy or surgical resection.



19.24. Glioblastoma multiforme.

Meningiomas

Meningiomas account for 15% of intracranial tumours. They commonly occur in the fourth to sixth decades of life, and have a slight female preponderance. The tumour is thought to arise from the arachnoid villi. Microscopically, they appear as whorls of fibroblasts around a central hyaline material which eventually calcify and form psammoma bodies (Fig. 19.25).

Malignant tumours exist and are defined by frequency of mitosis, invasion of the brain, and metastases. The common sites are the parasagittal (Fig. 19.26), sphenoid wing, and olfactory groove regions. The tumour tends to produce hyperostosis of the skull.

Meningiomas are generally benign, and their clinical course is usually long. The symptoms and signs are related to those of an intracranial mass lesions or seizures. CT scan can accurately diagnose the lesion. Complete surgical removal usually results in cure.

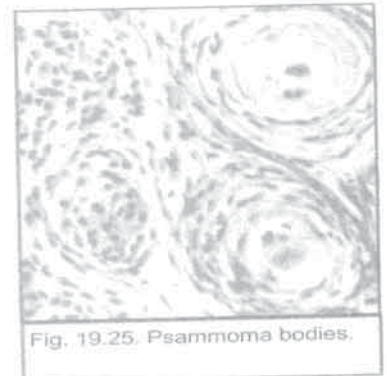


Fig. 19.25. Psammoma bodies.

Pituitary tumours

Pituitary tumours are broadly classified into non-functioning and functioning adenomas.

Non-functioning pituitary adenomas

1. These account for about 30% of pituitary tumours and are often seen in the fourth and fifth decades of life. They have also been referred to as null cell tumours, undifferentiated tumours, and non-hormone producing adenomas.
2. Because they are non-functioning, they are not generally diagnosed until they are very large. Their presentation is by optic chiasma compression that causes visual field deficits.
3. The usual treatment is microscopic trans-sphenoidal excision.

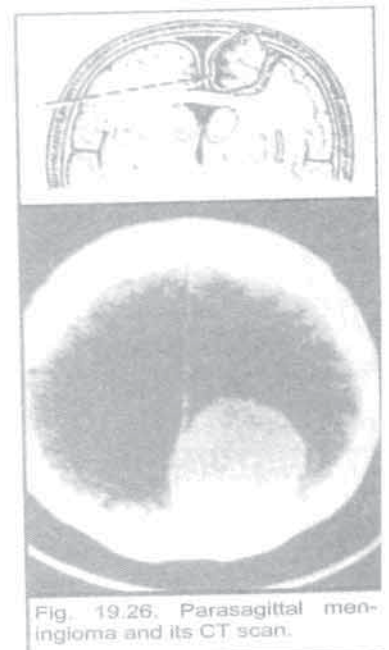


Fig. 19.26. Parasagittal meningioma and its CT scan.

Functioning pituitary adenomas

- Pituitary adenomas are classified according to the hormones they secrete. They include
 - Prolactin-secreting adenomas.
 - Growth hormone-secreting adenomas that produce acromegaly or gigantism.
 - Glycoprotein-secreting adenomas that produce excess amounts of TSH, LH, or FSH.
 - ACTH-secreting adenomas that produce Cushing's disease.
 - Some of these secrete more than one hormone, e.g., prolactin growth hormone-secreting adenomas.
- Adenomas may be further divided according to microadenomas that are less than 1 cm in size and macroadenomas that have a larger size.
- Functioning adenomas are diagnosed by clinical changes, hormone assessment, and radiology, namely MRI and CT scan.
- Prolactinomas represent 40% of all pituitary adenomas. They cause amenorrhoea, galactorrhoea, and infertility in women, while in men they cause impotence or may be asymptomatic. It should be noted that hyperprolactinaemia in the presence of a macroadenoma does not necessarily mean that the tumour is a prolactinoma. Mild prolactin elevation (less than 150 ng/mL) can be due to pituitary stalk compression.
- Except for specific situations, bromocriptin (anti-prolactin drug) has virtually replaced surgery as the treatment of choice for prolactinomas.
- The trans-sphenoidal route, whether sublabial or transnasal, is preferred for excision of pituitary adenomas. In case of an invasive tumour with incomplete excision radiotherapy is required.

FACE, LIPS AND PALATE

Development of the face, lips and palate

Development of the face (Fig. 20.1)

The human face is formed by fusion of five embryonic prominences. Each process is formed of a core of mesenchymal tissue lined by epithelium of endodermal origin and covered by surface ectoderm. They encircle the stomodaeum (primitive mouth).

- One frontonasal process. The frontonasal process (made of proliferation of neural crest from the forebrain) is indented by two olfactory pits dividing it into a median and two lateral processes.
- Two maxillary processes.
- Two mandibular processes. These will form the floor of the mouth, lower jaw, lower lip and the two superior portions which give rise to the maxillary process.

External ear

The external auditory canal is formed from the first branchial groove while the auricle is derived mainly from the second branchial arch from six tubercles.

Development of the lips (Fig. 20.1)

Upper lip

- The lateral parts are formed of the maxillary process below the nostrils.
- The middle part of upper lip (philtrum) is formed of the median part of frontonasal process.

Lower lip

The lower lip is formed by fusion of the two mandibular processes.

Development of the palate

- **Primary palate.** This is the anterior part of the palate and is also called the premaxilla. It is the part that carries the four incisor teeth. The premaxilla is derived from the median part of the frontonasal process (the same part that forms the philtrum of upper lip).
- **Secondary palate.** From each maxillary process a palatal process grows medial across the dorsum of the tongue. The two palatal processes unite with each other and with the premaxilla from before backwards together with the nasal septum thus separating the nasal cavities from each other and from the oral cavity.

CHAPTER CONTENT

- Development of the face, lips and palate
- Congenital anomalies
- Maxillo facial injuries
- Infections of the face
- Lip cancer

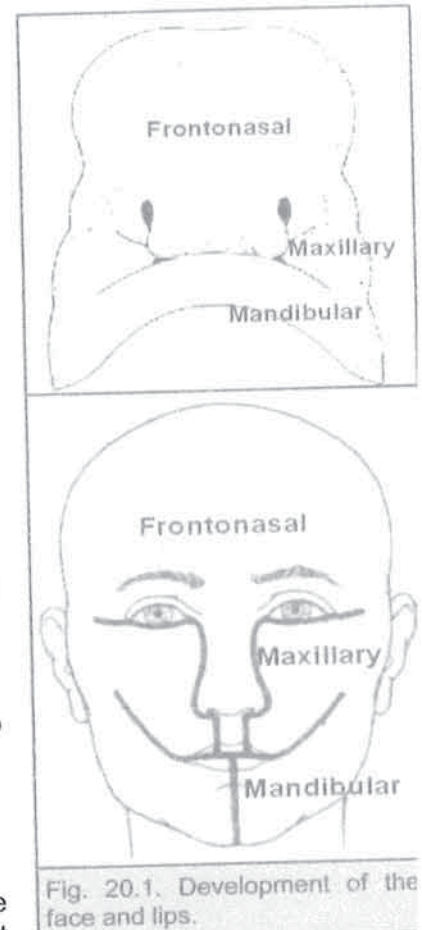


Fig. 20.1. Development of the face and lips.

Congenital anomalies

The common anomalies are

1. Cleft lip.
2. Cleft palate.
3. Preauricular sinus. This is due to imperfect fusion of the auricular tubercles. When occluded a cyst is prone to develop and might develop into an abscess, if it bursts a resistant ulcer will result.
4. Dermoid cysts. Sequestration dermoid cysts occur at the lines of embryonic fusion. The most frequent of which is the external angular dermoid (chapter 12).
5. Pierre Robin syndrome consists of cleft palate associated with receding mandible (micrognathia) and posterior displacement of the tongue obstructing the oropharyngeal airway.
6. Mandibular prognathism which means protrusion of the mandible.

Clefts of the lip and palate are the most frequent congenital anomalies of the head (1700 live births). The incidence is less in black and oriental races. Therefore, they will be discussed in details.

Cleft lip (hare lip)

Aetiology

The possible aetiological factors include

1. Familial disease due to genetic susceptibility.
2. Consanguinity.
3. Prenatal exposure to alcohol, anticonvulsants, x-ray or virus infection as German measles in the first 3 months of pregnancy.

Pathology

- Cleft lip is caused by failure of fusion between the median part of the frontonasal process and one (unilateral) or both (bilateral) lateral maxillary processes in the developing face.
- The condition may be unilateral (Fig. 20.2) (most common on to the left side) or bilateral (Fig. 20.3).
- The cleft can be complete (reaching the nostril floor) or incomplete. With complete lip clefts the orbicularis oris muscle is completely interrupted. This leads to flaring and flatness of the nares on the affected side (Fig. 20.4).
- There is lack of continuity of the skin, mucous membrane and orbicularis oris across the cleft.
- The cleft lip may be simple or complicated if it is associated with cleft palate or alveolus.
- There is short lip-nose distance on the affected side.

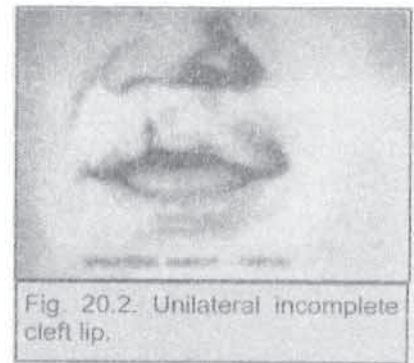


Fig. 20.2. Unilateral incomplete cleft lip.



Fig. 20.3. Bilateral cleft lip

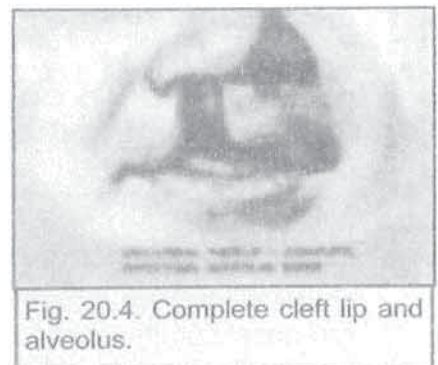


Fig. 20.4. Complete cleft lip and alveolus.

- Cleft lip does not interfere with suckling, but there might be associated abnormal teeth growth.
- Cleft lip may be associated with other congenital anomalies in up to 35% of cases (cleft palate, velopharyngeal incompetence, coloboma, microphthalmia, encephalocele, ear tags or torticollis).

Treatment

- Surgery is the only treatment
- Timing. Operation is best performed at the age of 3-6 months.
- Pre-requisites. The infant should be at least 10 pounds in weight and the haemoglobin level should be at least 10 gm%.
- Aim of surgery. The aim of surgery is to improve appearance. There is no functional loss.
- Principles of the operation (Fig. 20.5)
 - Paring of the edges.
 - Repairing the defect by suturing the three layers of the lip (skin, muscle and mucous membrane) taking care to adjust the vermillion and mucous borders. The sutures are not made in a straight line but in a zigzag way to avoid notching of the lip margin as the scar contracts.

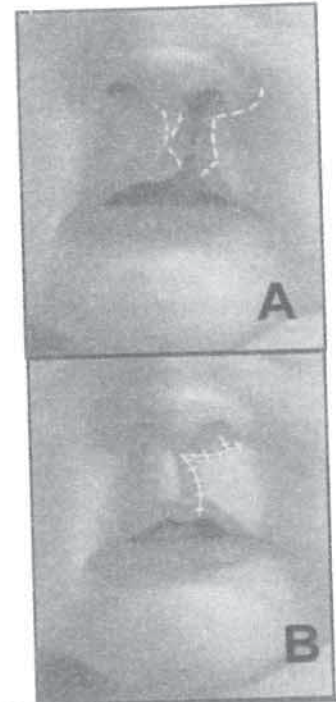


Fig. 20.5. Repair of cleft lip.

Cleft palate

Isolated cleft palate forms one third of all facial clefts.

Aetiology

Predisposing factors are as mentioned for cleft lip.

Pathology

Cleft palate is caused by arrest of fusion between the two palatal processes, and possibly with the premaxilla.

Types (Fig. 20.6)

1. Cleft uvula.
2. Cleft soft palate.
3. Cleft soft and hard palate (complete).
4. Complete cleft palate plus one side of premaxilla (bipartite).
5. Complete cleft palate plus both sides of premaxilla (tripartite, Fig. 20.7).



Fig. 20.6. Types of cleft palate

- A. Cleft soft palate
- B. Cleft soft and hard palate
- C. Complete cleft palate plus one side of premaxilla (bipartite)
- D. Complete cleft both sides of (tripartite)

Effects on function

- (a) Impairment of normal suckling, due to inability to create a negative intra-oral pressure because of the oro-nasal communication
- (b) Food will reflux into the nose and may be aspirated leading to aspiration pneumonia

- (c) Inadequate emptying of the middle ear due to abnormal levator palati insertion, preventing adequate aeration of the Eustachian tube thus predisposing to recurrent otitis media. This may lead to hearing loss.
- (d) Speech defects secondary to.
 - a. Inadequate velopharyngeal mechanism. Opposition of velum (the muscles of the soft palate) against the pharyngeal wall to separate the oral from the nasal cavity) is impaired. Nasal tone due to naso-oral communication.
 - b. Hearing loss
- (e) Distortion of facial growth.
- (f) Interference with normal teeth alignment.

Treatment

- Timing of operation 12-18 months.
- Preoperative management
 - Attention to feeding. As there is inefficient breast feeding, a bottle with a large hole is used or spoon feeding is practiced in an upright position.
 - Prevention and treatment of chest infection.
- Objectives of surgery
 - Closure of oro-nasal communication
 - Achieving a competent velopharyngeal sphincter.
- Principles of surgery (Fig. 20.8)
 - Paring of edges.
 - Suturing is done in 3 layers in the middle line (nasal mucosa, muscle layer then oral mucosa).
 - Lateral relaxation incisions are needed.
 - Fracture of the pterygoid hamulus to relax the tensor palati.
- Post operative treatment
 - Speech therapy.
 - Orthodontic treatment.

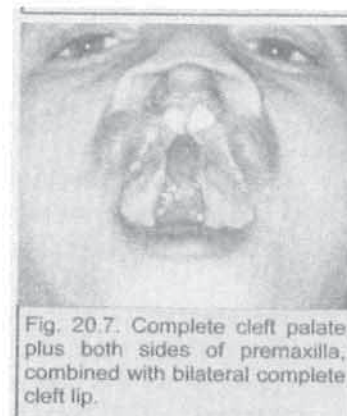


Fig. 20.7. Complete cleft palate plus both sides of premaxilla, combined with bilateral complete cleft lip.

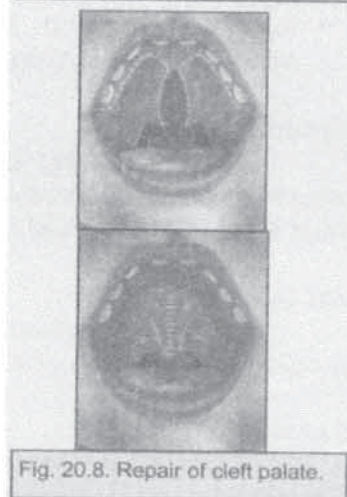


Fig. 20.8. Repair of cleft palate.

Maxillofacial injuries

Maxillofacial injuries are frequent with road traffic accidents, fights, and contact sports as soccer.

In treatment of patients with extensive maxillofacial injuries, the order of priorities is consistent

1. Patent airway. The patient may have compromise of his respiration due to blood, dentures or vomitus obstructing the upper airway. The usual measures to keep a patent airway are followed (chapter 2). Tracheostomy is rarely indicated.
2. Effective breathing.
3. Control of haemorrhage by direct pressure or ligation of the bleeding vessels. If the patient is shocked, suspect associated abdominal or chest injuries. Remember that isolated facial injuries rarely cause shock.

Soft tissue injuries

Skin wounds

These should be treated in theatre under sterile conditions:

- Clean and irrigate the wound.
- Remove foreign bodies.
- Minimal wound debridement.
- Cut wounds and lacerations are sutured.
- Avulsed flaps are sutured back after ensuring vascularity at the tip.
- Local flaps or skin grafts are needed if there are skin defects.

Facial nerve injuries

These can be diagnosed on physical examination.

- If the injury is medial to the midpupillary line, it requires no treatment.
- Lateral injury requires nerve repair under magnification (chapter 18).

Parotid injury

- Injury of the duct requires end to end anastomosis over a small silastic catheter. If the injury is at the distal part of the duct, the proximal cut end is sutured to the oral mucosa.
- Injury to the gland. The skin is sutured and a small drain, is inserted. There will be minor salivary leakage which usually stops in 3 weeks.

Eyelid injuries

Careful suturing of all layers of the lid. Repair of cut levator should be done, otherwise ptosis will result. The tarsus should be repaired. Injury to the lacrimal apparatus must be recognized and repaired to avoid epiphora and dacryocystitis.

Ear injuries

Full thickness tears are sutured by cutaneous perichondrial sutures. Haematomas should be evacuated otherwise cauliflower ear will result.

Nasal injuries

Nostril tears should be sutured carefully in two layers. Septal haematoma should be evacuated to avoid septal cartilage resorption which will end in a saddle nose deformity.

Lips

These should be sutured in 3 layers with respect to the anatomical landmarks.

Intra oral injuries

The edges are debrided and loosely approximated.

Animal bites

These are usually heavily contaminated. Treatment consists of prompt excision, antibiotics and rabies vaccination. The wound is left open.

Fractures of facial bones

These fractures may be closed or open.

Mandibular fractures**Aetiology**

Falls, kicks, fist blows, car accidents or pathological.

Site

- Fractures may occur in the symphysis, body, angle, ramus, condyles, coronoid alveolar process.

- Fractures of the body are the commonest and usually occur close to the mental foramen, where the bone is weakened by the marked curvature and by the deep canine socket. In bilateral cases, the digastric and geniohyoid muscles pull the chin fragment and the attached tongue backwards, impairing the airway. Fractures of the angle are minimally displaced as they are splinted by the masseter and the pterygoid muscles.

Clinical features

Symptoms

- Pain, especially on attempts to open the mouth.
- Blood-stained saliva. Fractures of the mandible are usually compound into the mouth because the mucoperiosteum is firmly attached to the bone.
- Impairment of speech and swallowing.
- Sometimes there is anaesthesia of the lower lip.

Signs

- Swelling and haematoma in the floor of mouth (Coleman's sign).
- Local tenderness.
- Crepitus
- Irregularity of the line of teeth (Fig. 20.9).

Investigations

Plain X-ray is diagnostic. A panoramic view shows the whole mandible (Fig. 20.10).

CT may be needed.

Treatment

1. First-aid treatment. The jaw is supported with 4-tailed bandage and analgesics are prescribed for pain. Antibiotics and mouth hygiene are important to prevent infection.
2. Reduction and fixation. If there is displacement, reduction is done under anaesthesia. Fixation (for 3-6 weeks) is done by
 - a. Arch bars or inter-dental wiring. In the latter the patient's jaws are wired together (Fig. 20.11) and a liquid diet is given. This technique is appropriate for a majority of lower jaw fractures.
 - b. Plate and screws. More complicated fractures require open reduction through an incision and internal fixation of bone ends by plates and screws (Fig. 20.11).

Fractures of the Maxilla

Aetiology

This usually occurs secondary to car accidents.



Fig. 20.9. Irregularity of the line of teeth in a case of mandible fracture



Fig. 20. 10. A panoramic view shows the whole mandible. In this case the fracture is situated near the mental foramen and has been fixed by plates and screws.

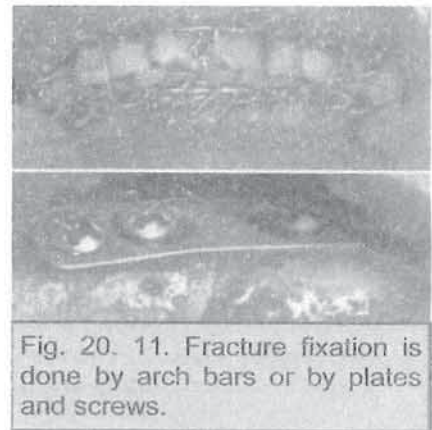


Fig. 20. 11. Fracture fixation is done by arch bars or by plates and screws.

Clinical features

There is pain, excess salivation, mal-occlusion, epistaxis, diplopia, swelling, and crepitation. The deformity can be visualized by X-ray or CT scan (Fig. 20.12).

Classification

Le Fort classified maxillary fractures into three varieties (Fig. 20.13)

- **Le Fort I.** This is a transverse fracture above the level of the teeth. It is treated by intermaxillary fixation which in turn is fixed to the inferior orbital margin by wires.
- **Le Fort II.** This is a pyramidal fracture, traversing the base of the nose through the posterior wall of maxillary antrum and across the orbit. It is treated by intermaxillary fixation fixed by wires to the zygomatic process of frontal bone.
- **Le Fort III.** There is craniofacial disjunction, i.e., separation of the facial bones from their cranial attachment. Treatment includes correction of nasal and zygomatic fractures and treatment of fracture of the maxilla as in Le Fort II.



Fig. 20.12. CT scan showing a depressed fracture of anterior wall of left maxillary antrum.



Fig. 20.13. Le Fort classification of maxillary fractures.

Fractures of the nose

Clinical features. There is pain, swelling, epistaxis, crepitus. Plain X-ray is diagnostic.

Treatment is by digital or instrumental manipulation to reduce the fracture. The position is then fixed by intranasal packing for 3 days with an external splint for 7 days. Neglected old fractures require osteotomies.

Fractures of the zygoma

Clinical features include pain, swelling in eyelids, flat cheek, numbness in the cheek and upper teeth due to injury of the infraorbital nerve, and crepitus with irregular infraorbital margin.

Blowout fractures

These are depressed fractures of the orbital floor with herniation of some of the orbital contents into the maxillary sinus due to sudden increase in the intraorbital pressure. blow by a fist is a common cause.

Clinical features. The patient complains of diplopia and limited up and down gaze with or without enophthalmos.

Temporomandibular dislocation

Aetiology

- Direct blows.
- Yawning.
- Wide opening of the mouth under anaesthesia. It is commonly bilateral and affect middle aged females.

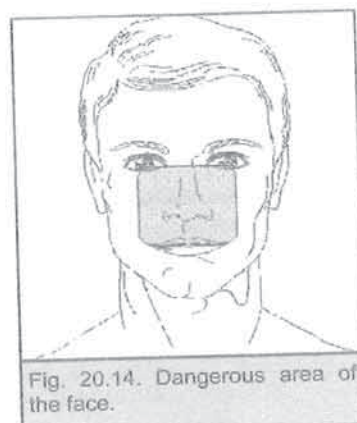


Fig. 20.14. Dangerous area of the face.

Clinical features

There is pain and dysarthria. The mouth is held open with fixed jaws. In unilateral cases, the chin is deviated to the opposite side.

Treatment

- Reduction (better under anaesthesia) by downward traction on the molars with the padded thumb, together with upward rotation of the body with the outside fingers.
- In recurrent cases excision of the meniscus is done.

Infections of the face

Boils or carbuncles can develop in the face. The area of the face between the lines passing from the outer canthus to the angle of the mouth is called the "dangerous area" because infection in this area is liable to cause cavernous sinus thrombosis (Fig. 20.14).

Routes of spread to cavernous sinus

1. The angular vein communicates with the ophthalmic veins which drain into the cavernous sinus.
2. The anterior facial vein communicates with the pterygoid venous plexus in the infratemporal fossa through the deep facial vein. The pterygoid venous plexus is connected to the cavernous sinus by an emissary vein that passes through the foramen ovale at the skull base.

Lip cancer

Epithelioma (squamous cell carcinoma) is the commonest lip malignancy (see chapter 12).

Aetiology

1. Prolonged exposure to the ultraviolet rays of the sun.
2. Continuous irritation and hyperplasia due to cigarette smoking or the use of hot tobacco pipes.

Pathology

Histological picture

Squamous cell carcinoma, which is usually well-differentiated.

Gross picture

The lower lip is more affected than the upper. The lesion usually starts as a nodule or erosion which resists treatment. Later the typical ulcer (Fig. 20.15) becomes evident.

- Raised everted edges.
- Indurated base and margin (induration extends beyond the edge).
- Possibly spread to cervical lymph nodes (Fig. 20.16).
 - The central part of lower lip drains to submental nodes. The lateral parts drain to submandibular nodes.
 - Later upper deep cervical nodes are involved.

Treatment

Treatment follows the principles outlined in chapter 12.

Primary tumour

Surgery. Excision should include the lesion with a safety margin.

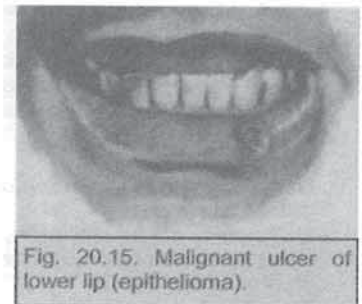


Fig. 20.15. Malignant ulcer of lower lip (epithelioma).

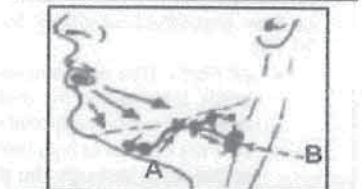


Fig. 20.16. Lymphatic spread of lower lip cancer. A. Submandibular nodes B. Upper

- For lesions involving up to one third of the lip surgical treatment can be accomplished by "V" shaped excision and primary suture in 3 layers; mucosa, muscle, and skin.
- For bigger lesions plastic reconstruction will be needed.

Radiotherapy is a good alternative because squamous cell carcinoma is radio-sensitive.

Lymph nodes

If there are lymph node metastases, a suprahyoid or a complete block dissection

Prognosis

Epithelioma of the lip has a better prognosis than that of the cheek, tongue or floor of mouth.

MOUTH, CHEEK AND TONGUE

The oral cavity is full of commensals (streptococci, staphylococci, and others). They cause severe infection in the wounds caused by human bite, but their ability to cause harm to the oral cavity is reduced by the following:

1. Regular desquamation and rapid replacement of surface cells.
2. Saliva that washes the oral cavity continuously.
3. The mild antibiotic activity of saliva.

CHAPTER CONTENTS

- Stomatitis
- Cysts of the floor or mouth
- Developmental anomalies of the tongue
- Tongue injuries
- Glossitis
- Tongue ulcers
- Carcinoma of the tongue
- Carcinoma of the cheek

Stomatitis

This is a group of inflammatory, erosive, and ulcerative conditions that affect the mucous membrane of the oral cavity.

Predisposing factors

1. Anaemia and VB12 deficiency that lead to thin, atrophic epithelia and loss of tongue papillae.
2. Immunodeficiency and autoimmune diseases.

Types

1. **Aphthous stomatitis.** Painful ulcers up to 0.5 cm across, round or oval with a yellow base and red margin (Fig. 21.1). They heal normally within 10-14 days.
2. **Herpes simplex infections.** Small vesicles appear and rapidly break down to form small, yellow ulcers with bright red margins. They occur on the gingiva, cheeks, lips, and tongue. Skin of the cheek and around nostrils can be also affected. The patient is unwell, febrile with enlarged submandibular lymph nodes.
3. **Monilial stomatitis.** This is a fungus infection by candida species characterized by formation of white membranous lesions. It occurs in debilitated, chronically ill patients under cytotoxic drugs, cortisone therapy or prolonged treatment by broad-spectrum antibiotics. It affects mainly the tongue. Treatment is by gentian violet and amphotericin-B lozenges.

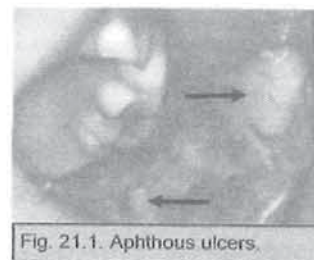


Fig. 21.1. Aphthous ulcers.



Fig. 21.2. Ranula.

Cysts of the floor of mouth

Mucous cysts of minor salivary glands

These are either retention cysts behind minute calculi or extravasation cysts. They form a soft pinkish or bluish swellings up to 1.5cm in diameter Treatment is excision.

Ranula

This is a retention cyst arising from a sublingual salivary gland. Saliva distends the cyst in the floor of the mouth.

Pathology. The wall is composed of a thin fibrous capsule, which is lined by macrophages. It may contain gelatinous material. If it extends down into the neck over the posterior margin of the mylohyoid (diaphragm of the mouth), it forms a plunging ranula. It may rupture but usually refills again.

Clinical picture. A ranula forms a translucent, bluish, cystic swelling (Fig. 21.2), with prominent blood vessels running over its surface together with stretched submandibular duct.

Treatment. Excision of the sublingual gland with the cyst is very difficult (as the wall is thin and adherent). Partial excision of the roof, the edges of which are sutured to the mucosal lining the floor of the mouth (marsupialisation) is the treatment of choice.

Lingual and sublingual dermoid

Opaque swellings lined by stratified squamous epithelium. The cysts are filled with mucous (fluctuant), or with a doughy mass of keratin. They are found in the midline of the tongue, or in the floor of the mouth (Fig. 21.3), either in the midline (the commonest type), or laterally in the submandibular region. Although congenital, sublingual dermoids usually appear after puberty.

Treatment is excision through the floor of the mouth, but a submandibular incision can be used for large ones.

Developmental anomalies of the tongue

Tongue tie

It is due to short fibrous lingual frenum (Fig. 21.4). It causes impairment of tongue movements, and possibly speech defects. Treatment is by division of the frenum below the undersurface of the tongue.

Macroglossia

Persistent, painless enlargement of the tongue.

Congenital causes

1. Cavernous haemangioma.
2. Congenital arteriovenous fistula.
3. Lymphangioma.
4. Neurofibromatosis.

Acquired causes

1. Cretinism.
2. Acromegaly.
3. Amyloidosis.

Tongue injuries

Aetiology

Biting the tongue is the commonest cause. Epileptic patients may bite their own tongues during attacks. Tongue lacerations can be associated with jaw fractures due to road traffic accidents. Bleeding (as a result of damage to the lingual artery) may be very serious if the patient is unconscious as it can endanger the airway.



Fig. 21.3. Sublingual dermoid cyst.



Fig. 21.4. Tongue tie.

Treatment

Bleeding is arrested by hooking the tongue forwards with a finger and compressing it against the mandible. Then the lacerations could be sutured in the operating room. Haematoma of the tongue producing respiratory embarrassment may require tracheostomy.

Inflammation of the tongue (glossitis)

Chronic superficial glossitis

Aetiology

This is due to chronic irritation (smoking, sharp tooth, sepsis, syphilis, spirits, and spices). It occurs in middle and old ages.

Pathology

- The disease affects the anterior two thirds of the tongue.
- Hypertrophy of the papillae produces red hyperaemic patches.
- Overgrowth of the epithelium makes it thickened, indurated and opaque so that the red patches are replaced by white ones, and the tongue appears as if it were coated with white paint. This constitutes leukoplakia (Fig. 21. 5 & 6).
- In the next stage the epithelium may be shed over a considerable area producing a red glazed tongue.
- Submucous fibrosis occurs causing ulcers, fissures and warty projections.
- Chronic superficial glossitis is precancerous (Fig. 21.6).

Treatment

1. Elimination of the irritating source.
2. Mouthwash.
3. In resistant cases excision of the lesion.

Candida fungus infectin of the tongue occurs due to prolonged use of antibiotics (Fig. 21.7).

Tongue ulcers

Only common types of tongue ulcers will be discussed.

1. Dental ulcer

This is due to repeated trauma by a carious and broken tooth or ill fitted denture. It occurs at the side of the tongue near the offending tooth, yet this relation might not be apparent when the tongue is fully protruded for inspection. The ulcer is small, oval or rounded with granulation tissues at the floor, soft base and sloping margins. It is painful with septic enlargement of the draining lymph nodes. In chronic cases the edges may be raised with indurated base, so biopsy is needed to exclude malignancy. Treatment is by removal of the offending cause and the use of an antiseptic mouthwash.



Fig. 21.5. Leukoplakia of chronic superficial glossitis.



Fig. 21.6. Three areas of malignant transformation on top of leukoplakia.

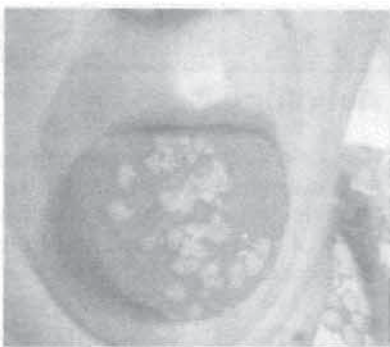


Fig. 21.7. Candida (monilia) glossitis.

2. Aphthous ulcers

The aetiology of these ulcers is not exactly known. There are multiple, small ulcers affecting all mucosal lining of the oral cavity. They are very painful, rounded, yellow ulcers near the tip of the tongue, with red margin, soft base and no lymph node enlargement. They normally heal, in 2 weeks. Treatment is by antiseptic mouth wash, alkaline lotion as 2% sodium bicarbonate and anaesthetic jelly.

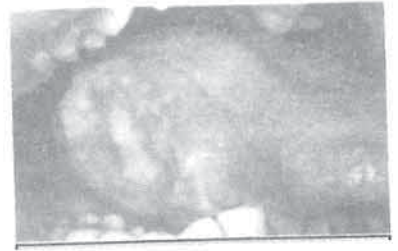


Fig. (21.8) Malignant tongue ulcer

3. Neoplastic ulcers

Usually squamous cell carcinoma.

Carcinoma of the tongue

Aetiology

- **Age incidence** is usually above 60 years.
- **Sex incidence** used to be M:F = 10:1. Nowadays, it is just slightly higher in males. The recent lowering of incidence in males is due to more efficient treatment of syphilis, improved standards of oral hygiene. The recent increased incidence in females is due to increase of smoking in females.
- **Predisposing factors.** Chronic irritation by smoking, sepsis, spices, spirits, sharp teeth, and bad oral hygiene.
- **Precancerous lesions**
 - Syphilis.
 - Dental ulcers.
 - Chronic superficial glossitis.
 - Papilloma of the tongue.
 - Leukoplakia (any white lesion which cannot be rubbed off). It shows hyperkeratosis and possibly dysplasia.
 - Erythroplakia. This is deep reddening with mucosal atrophy and leukoplakia.



Fig. 21.9. Tongue cancer showing as an elevated plaque.

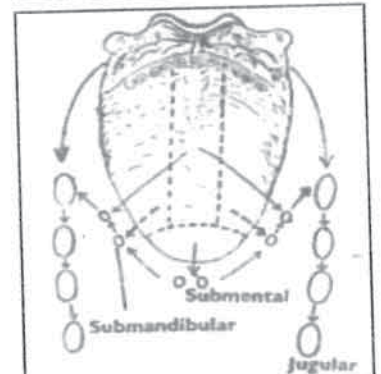


Fig. 21.10. Lymphatic spread of tongue cancer.

Pathology

Site

The commonest site of carcinoma of the tongue is the lateral margin of the anterior two thirds (50%). This is followed by the posterior third. Less common sites include the ventral and the dorsal surface. The tip is rarely affected.

Gross types

1. Malignant ulcer (Fig. 21.8) with deep irregular floor with necrotic tissues, raised nodular everted edges and a hard indurated base.
 2. Raised oval plaque (Fig. 21.9).
 3. Hard submucosal nodule (less common).
 4. Deep indurated fissure.
 5. Diffuse infiltrating tumour (wooden base) is rare.
- The surrounding mucous membrane may show leukoplakia.

Microscopic appearance

Squamous cell carcinoma contributes to 90% of malignant lesions of the tongue. Tumours of the posterior third are usually less differentiated than those of anterior two thirds of tongue. Rarely tumours include basal cell carcinoma and adenocarcinoma from the minor salivary glands.

Carcinoma in situ, means evidence of malignant cells without invasion of the basement membrane. At this stage there is neither lymphatic nor blood spread.

Spread

1. Direct spread. Tumours of the anterior two thirds start laterally and may reach the floor of the mouth before they cross the midline. They may invade the mandible. Tumours of the posterior third spread to the tonsils, pharyngeal wall and larynx. They may cross to the other side of the tongue.
2. Lymphatic spread. Unlike squamous carcinoma of the lip, carcinoma of the tongue disseminates early to the lymph nodes of the neck (Fig. 21. 10).
 - Spread from the tip of the tongue to the submental then bilaterally to submandibular and upper deep cervical nodes.
 - Tumours of the lateral third disseminate to the ipsilateral submandibular and then to the upper deep cervical lymph nodes. Those near the midline disseminate bilaterally.
 - Posterior third. Spread occurs directly to the upper deep cervical lymph nodes.
3. Blood spread is a rare event. It is more likely for tumours of the posterior third.

TNM staging system is used to describe the extent of the tumour (table 21.1).

T ₀	No evidence of tumour
T _{is}	Carcinoma in situ
T ₁	<2 cm
T ₂	2-4 cm
T ₃	>4 cm
T ₄	Involvement of base
N ₀	No lymph nodes
N ₁	Ipsilateral single node <3 cm
N ₂	Ipsilateral or contralateral lymph nodes <6 cm
N ₃	Lymph nodes >6 cm
M ₀	No metastasis
M ₁	Distant metastasis

Complications

1. Inhalation of necrotic tissues, leading to bronchopneumonia.
2. Combined cancer cachexia and starvation due to pain and dysphagia.
3. Haemorrhage from erosion of the lingual artery or in tumours of posterior 1/3 erosion of the internal carotid artery.
4. Asphyxia may occur either due to pressure of enlarged fixed lymph nodes on the air passages or due to oedema of the glottis.

Clinical picture

The classic picture of tongue cancer is that of an old man sitting in the outpatient clinic with cotton wool in his ear, and blood stained saliva dribbling from the mouth.

Early cases

The disease is symptomless. The patient may complain of a persistent ulcer with indurated base and everted edges. There may be:

- Deep indurated fissure.
- Lobulated indurated mass with overlying yellow patches of submucosal necrosis.
- Oval raised papillated plaque with overlying white keratin fleck.

Late presentation

- Pain in tongue, first due to infection, later due to lingual nerve infiltration. Pain is referred to the ear or temple through the chorda tympani via the auriculo-temporal nerve. In tumours of the posterior third pain may be felt on swallowing.
- Salivation due to the pain plus restricted tongue mobility. It may be blood stained and smells badly (necrosis and infection).
- Inability to articulate clearly.
- Enlarged cervical lymph nodes.
- Ankyloglossia (tongue fixation) due to extensive infiltration of the floor of mouth.
- Picture of complications as cachexia, pneumonia and bleeding.

Investigations

1. Biopsy from the ulcer edge. Any ulcer or fissure-like lesion that resists treatment should be biopsied.
2. Fine needle aspiration cytology of suspected cervical lymph nodes.
3. CT of neck and mandible.

Treatment

Different modalities of treatment are available. Surgery and radiotherapy are the main lines of treatment. Chemotherapy is used as an adjuvant in some cases.

Radical treatment for early cases**Surgery****Indications**

1. Small growths. T₀, T₁ and T₂.
2. Incomplete regression or recurrence after radiotherapy.
3. Cancer on top of a precancerous lesion as leukoplakia.
4. Presence of the tumour very close to the mandible or infiltrating it.

Preoperative preparation

Care of teeth and oral hygiene. Preoperative irradiation by 4000 rad may be advised.

Resection procedures

- Carcinoma in situ needs excision with 1 cm safety margin on the sides and 0.5 cm safety margin in depth. The defect is closed by advancing a mucosal flap from the floor of mouth.
- Carcinoma of the anterior two thirds of the tongue is excised with 1.5 cm safety margin. This may amount to partial glossectomy, hemiglossectomy or near total glossectomy.
- Carcinoma of the posterior third is treated either by total glossectomy which is difficult due to difficult access (median mandibulotomy will facilitate oral resection) or by external irradiation.
- If there are lymph nodes metastases, they are excised by complete neck dissection. If the lymph nodes are bilateral, a complete dissection is performed on the more affected side and a selective neck dissection on the less affected side preserving the internal jugular vein (see Chapter 24).

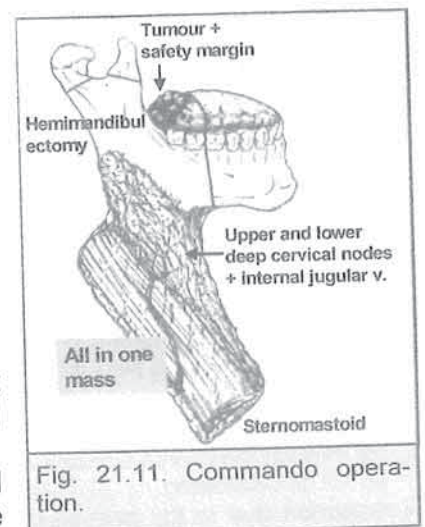


Fig. 21.11. Commando operation.

- If the mandible is affected, it must be excised together with the tongue and the affected lymph nodes. Combined mandibulectomy and neck dissection operation is known as **Commando operation** (Fig. 21.11).
- To close the huge defects following major resectional operations on the tongue, floor of the mouth and the mandible, various plastic procedures as pectoralis major myocutaneous flap are available.

Radiotherapy

Indications

T₁ and T₂ (less than 4 cm) may equally benefit from surgery or radiotherapy. The advantages of radiotherapy is avoiding the disfiguring side effects of surgery. Its disadvantages are mucositis, dysphagia and osteoradionecrosis.

Irradiation can be performed by caesium or iridium needles or by external beam radiotherapy.

Palliation for advanced cases

Indications

1. Unresectable primary growth.
2. Fixed lymph nodes in the neck.
3. Distant metastases.

Methods

1. Radiotherapy.
2. Palliative resection of the primary if possible may comfort the patient.
3. Analgesics, nasogastric feeding or tracheostomy may be required.
4. Chemotherapy.

Prognosis (prognostic factors)

1. TNM staging. Lymph node involvement is the most important prognostic index.
2. The degree of tumour differentiation. Poorly differentiated tumours develop local recurrence even if surgery and radiotherapy are combined.
3. Extension of the tumour posteriorly to the oropharynx carries bad prognosis.
4. Combined surgery and radiotherapy improve prognosis.
5. Prognosis is better in females than males.

Carcinoma of the cheek

- **Aetiology.** Predisposing and premalignant conditions are similar to those of the tongue.
- **Pathology.** The commonest type is squamous cell carcinoma. Much less common is adenocarcinoma of minor salivary glands.
- **Clinical picture.** It usually presents by an ulcerated mass with raised everted edges and an indurated nodular floor (Fig. 21.13).
- **Treatment.** Interstitial or external beam radiation is the treatment of choice. Surgery is indicated for recurrent or residual tumours followed by reconstruction (as for the tongue cancer). Neck nodes as long as they are mobile are treated by neck dissection.

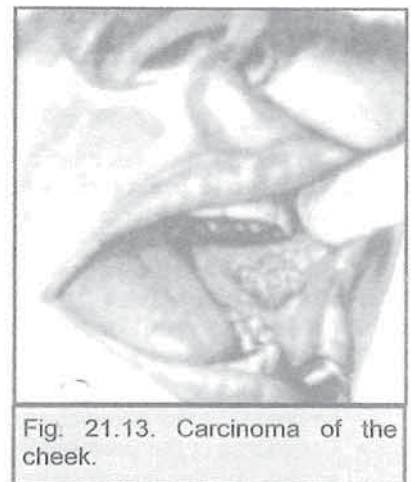


Fig. 21.13. Carcinoma of the cheek.

TEETH, GUMS AND JAWS

Development and structure of teeth

Development of teeth (Fig. 22.1)

The teeth start developing early in intrauterine life.

1. They arise from the primary dental plate, which is a ridge of ectoderm.
2. The plate dips in the mesoderm at the sites of the future teeth and is segmented to form the "enamel germs".
3. Each germ becomes further invaginated by mesodermal papilla and becomes surrounded by a fibrous vascular tissue to form the dental follicle.
4. In each follicle the enamel germ gives the ameloblasts which lay down the enamel. The mesodermal papilla gives rise to the dentine, cement and tooth pulp. The paradental epithelial debris of Malassez are fragments of ectoderm between the follicles.

CHAPTER CONTENTS

- Development and structure of teeth
- Related infections
- Classification of jaw swellings
- Epulides
- Odontomes
- Bone tumours of the jaws

Structure of the tooth (Fig. 22.2)

- The tooth consists of two parts; the crown projecting beyond the gum and the root embedded in the alveolus of the bone.
- It has a central cavity called the pulp which is rich in blood vessels and nerves.
- The pulp is surrounded by the dentine which is similar to bone but contains no cells.
- The enamel covers the crown while the cementine covers the dentine of the root. The cementine is surrounded by the dental periosteum (Periodontal membrane).
- The gum is formed of mucous membrane that is firmly attached to the periosteum at the alveolar margin of the bone.



Fig. 22.1. Teeth development.

1. Dental plate.
2. Enamel germs.
3. Dental follicles.

Related infections

Alveolar abscess

The abscess may result from the spread of infection from necrotic pulp into the periapical tissue. Pus may burst under the periosteum to form a subperiosteal abscess, which points externally or in the maxillary antrum. Infection may spread to the cavernous sinus.

Clinical features

- Marked toxæmia.
- Severe pain and cheek swelling with gum inflammation.
- Regional lymph nodes are enlarged and tender.

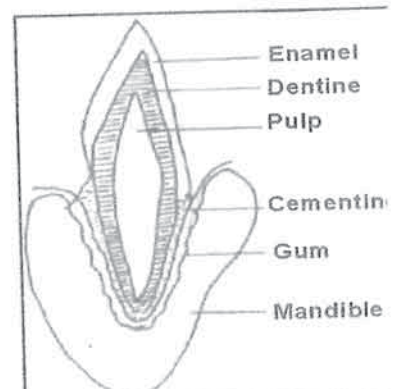


Fig. 22.2. Tooth structure.

Treatment

- Antibiotics.
- Extraction of the offending tooth to provide drainage.
- Incision and drainage of the subperiosteal abscess.

Osteomyelitis**Acute osteomyelitis**

Acute osteomyelitis usually affects the lower jaw as a complication of an alveolar abscess. The commonest organism is *Staphylococcus aureus*. The disease usually turns into the chronic type.

Clinical features. Clinically there is history of alveolar abscess followed by pain, fever and swelling. Trismus may be present.

Treatment. Antibiotics and mouth wash.

Chronic osteomyelitis

This usually affects the lower jaw because it has a single arterial blood supply, which passes near and parallel to the edge, in contrast to the upper jaw where the vessels pass at right angles to the jaw.

Aetiology

1. Following acute osteomyelitis.
2. As a complication of a compound fracture of the mandible.
3. Blood borne (rare).
4. Chemical necrosis due to substances used in industry, e.g., phosphorus, arsenic and mercury.
5. Radium necrosis, if irradiation is used for tumours nearby or infiltrating the jaw.
6. Rare causes include tuberculosis, syphilis, and actinomycosis.

Pathology

Infection starts in the medulla and spreads to the subperiosteal space. In general, it is similar to osteomyelitis in other bones. However differences include

- Involucrum is poor.
- Sequestra take a long time to separate.
- If the inferior dental artery is thrombosed a massive sequestrum results.

Clinical features

There is pain, fever and thickening of the jaw. This is followed by abscess and sinus formation. Trismus may be present if the molar region is involved.

Investigations

1. Plain X-ray will show the characteristics of chronic osteomyelitis which are bone necrosis (sequestrum) and new bone formation.
2. Culture and sensitivity of the discharged pus.

Treatment

1. Antibiotics and mouth hygiene.
2. Incision and drainage of an abscess, if present.
3. Saucerisation and sequestrectomy through an incision over the lower border of the mandible.

Classification of jaw swellings

Epulides = swellings of the mucoperiosteum or gum margin

1. Fibrous epulis.
2. Granulomatous epulis.
3. Myeloid epulis.
4. Sarcomatous epulis.
5. Carcinomatous epulis.

Odontomes = cysts or cystic tumours related to development of the teeth

1. Epithelial odontomes
 - a. Dental cyst.
 - b. Dentigerous cyst.
 - c. Adamantinoma (ameloblastoma).
2. Connective tissue and composite odontomes are not present in man.

Bone tumours

1. Benign
 - Osteoma.
 - Chondroma
 - Giant cell lesions of the jaw
2. Malignant
 - Osteogenic sarcoma.
 - Fibrosarcoma.
 - Secondaries.
 - Malignant tumours of the maxilla.

Epulides

An epulis is a solid swelling riding over the gum.

Fibrous epulis (Fig. 22.3)

This is a localized inflammatory hyperplasia of the gum submucosa due to chronic irritation. It usually occurs near the incisor teeth of the lower jaw. It consists of fibrous

tissue containing spindle cells arranged in whirly manner.

Clinical features. A small, painless, pedunculated swelling arises between two teeth. It is covered by intact mucosa. At first, it is soft and vascular. Later, it becomes firm and more fibrous.

Treatment. Local excision is followed by recurrence. The correct treatment is to extract the teeth on either side of the tumour and to excise the tumour with a wide base of mucoperiosteum.

Granulomatous epulis (Fig. 22.4)

This is actually a mass of granulation tissue adjacent to a carious tooth or gingivitis. It forms a red, soft, lobulated 'mass over the jaw, which bleeds easily on touch because is devoid of epithelial cover.

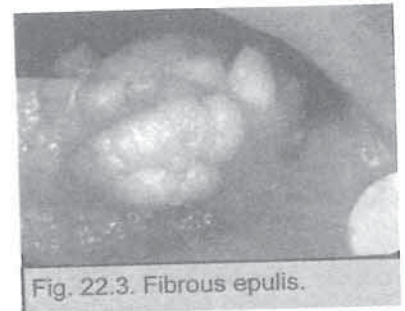


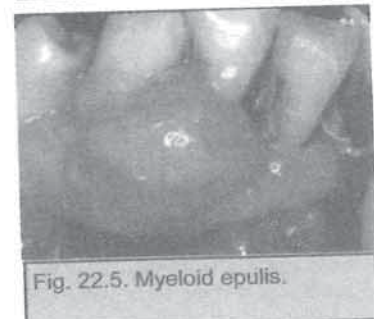
Fig. 22.3. Fibrous epulis.



Fig. 22.4. Granulomatous epulides.



Fig. 22.5. Myeloid epulis.



Treatment. Excision of the excess granulation tissue is done with diathermy, together with removal of the offending tooth.

Myeloid Epulis (Fig. 22.5)

This lesion affects the lower jaw more than the upper. It may arise from the inner osteoclastic layer of the periosteum, or it may arise from the alveolus itself. This explains the presence of osteoclasts in the tumour.

Pathology. This epulis forms a soft, lobulated, encapsulated swelling, with cystic areas in cut sections. Microscopically, there are spindle cells and giant cells together with fibroblasts and fibrous tissue.

Clinical features. The swelling is sessile, lobulated, bluish or brownish, and is covered with intact mucosa. It is fixed to the underlying bone. As it enlarges, the adjacent teeth are loosened and fall off. The mucous membrane is intact. X-ray of the jaw shows eating up of the bones.

Treatment. Wide excision with a safety margin.

Sarcomatous epulis

This is actually a parosteal fibrosarcoma arising from the outer layer of periosteum. It may occur on top of a fibrous epulis.

Clinical features. The tumour is sessile and fixed to the bone. It is of variable consistency. X-ray shows infiltration of the bones.

Treatment. Wide excision with the underlying bone; up to hemimandibulectomy.



Fig. 22.6. Dental cyst.

Carcinomatous epulis

This is an epithelioma arising from the mucous membrane covering the gum. The clinical picture and treatment are similar to carcinoma of the floor of mouth, but radiotherapy has no role.

Odontomes

Odontomes are cysts or cystic tumours related to the development of teeth. Only epithelial odontomes occur in man. The following are the types of odontomes.

Dental cyst

This cyst develops in connection with a pulpless infected tooth, which causes chronic irritation of the paradental epithelial debris of Malassez. It usually occurs in adult life.

Pathology

The cyst commonly affects the upper jaw. It is small, unilocular, slowly growing and expands the jaw on both sides. It is lined by squamous epithelium and contains glairy mucoid fluid rich in cholesterol crystals.

Clinical features

- A dental cyst presents as a painless, slowly growing swelling that usually arise in the anterior part of the upper jaw.

- As it expands the jaw, the bone becomes so thin that it gives a sensation of egg shell crackling when pressed by the examiner's finger. The nearby tooth is greyish (pulpless) and may be infected.
- X-ray shows expansion of the jaw around a well- defined cavity containing no teeth or trabeculae (Fig. 22.6).

Treatment

Extraction of the affected tooth is followed by opening of the cyst through an incision in the gum and enucleation of the cyst with removal of the lining membrane and fluid. The bone may be crushed to keep the shape of the alveolus.

Dentigerous cyst

This cyst occurs in children and adolescents at or after the second dentition in relation to an unerupted tooth (canine, premolar or third molar). It more commonly affects the lower than the upper jaw. It may be due to irritation of the paradental epithelial debris of Malassez or cystic degeneration of dental follicles.

Pathology

The cyst is slowly growing, expanding the jaw and may reach up to two inches in diameter. It is lined with squamous epithelium and contains glairy mucoid fluid rich in cholesterol crystals around an unerupted tooth.

Clinical features

- This is similar to dental cyst, but it usually occurs in the lower jaw near the angle of the mandible, and there is a missing tooth (unerupted).
- X-ray will show expanded jaw and a clear cyst with a tooth inside it (Fig. 22.7).

Treatment

Deroofing of the cyst is done with removal of the lining epithelium and fluid. The unerupted tooth is removed. The expanded jaw is crushed to restore its shape.

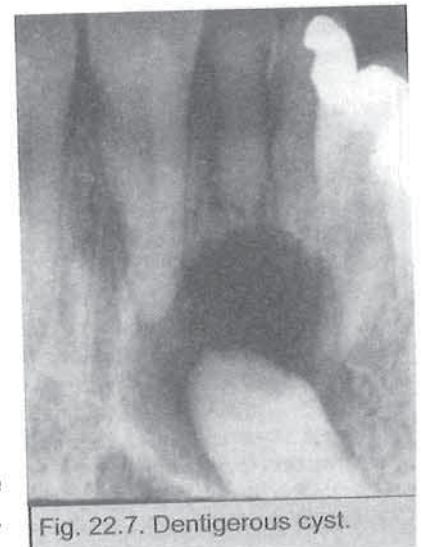


Fig. 22.7. Dentigerous cyst.

Adamantinoma (ameloblastoma)

Adamantinoma affects middle aged females (25-45 years). It occurs more commonly in the lower jaw. The cause is disputed; but it may be a basal cell carcinoma arising from the dental epithelium or from the paradental epithelial debris of Malassez.

Pathology

- Adamantinoma is a locally malignant tumour.
- It usually starts near the angle of the mandible and grows slowly both forwards in the body of the mandible and upwards in the ascending ramus.
- It is well encapsulated by fibrous tissue and expands the jaw.
- Trabeculae traverse the tumour dividing it into lobes and lobules. The cut section is pink or whitish with multiple cystic and solid areas.
- The cystic areas are lined by columnar cells and contain brownish mucoid fluid. The solid areas consist of fibrous tissue with particles of enamel. Osteoclasts are not present.

Complications

1. Ulceration, infection, bleeding and falling of teeth.
2. A pathological fracture is rare.
3. It is a locally malignant lesion, so it is liable to recurrence after inappropriate surgery. Sometimes, it turns malignant, (sarcoma from fibrous tissue element or carcinoma from the epithelial cells).

Clinical features

- There is a painless, lobulated, slowly growing swelling, usually in the lower jaw.
- It expands the jaw more on the outer than the inner side, so it is more obvious from the cheek than from the mouth.
- It is well defined and not tender.
- The overlying skin and mucous membrane are intact. The latter may ulcerate by the tumour or by displacement and loosening of the teeth.
- The swelling is bony hard in consistency, but egg shell crackling can be elicited over large tumours.
- No lymph nodes can be felt unless ulceration and secondary infection occur.

Investigations

1. Plain X-ray shows an expanding, translucent, well defined shadow, divided by bony septa into a more or less equalized lobules (fine soap bubble or honey comb appearance, Fig. 22.8).
2. CT scan and MRI.
3. Biopsy.

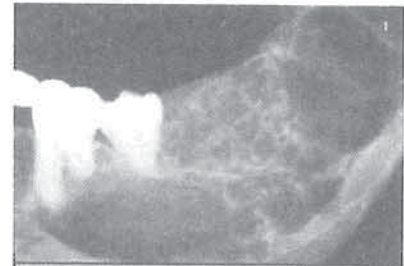


Fig. 22.8. Adamantinoma.

Treatment

Resection of the part of the mandible carrying the tumour, with a safety margin, which may amount to haemimandibulectomy. Reconstruction can be done either immediately or at a second stage by a bone graft (free or vascularized rib graft), or by a prosthesis.

Bone tumours of the jaws

Benign tumours and tumour-like conditions

Osteoma

The tumour may be of an ivory or a cancellous variety. **Ivory** osteoma affects the bones which develop in membrane as the ascending ramus of the mandible, the outer surface of the maxilla, palate, antrum and orbit. It is treated by excision.

Cancellous osteoma affects the bones which develop from cartilage as the maxilla and the symphysis menti. Treatment is by excision.

Giant cell lesions of the jaw

Giant cell granuloma

This is like osteoclastoma in large bones, but is benign. It affects the lower jaw more commonly. It arises above the symphysis menti as a soft reddish mass and grows backwards in the body of the mandible, but not upwards in the ascending ramus. The cut section shows a lobulated tumour. Histologically, there are giant cells, spindle cells and numerous blood vessels. Histologically, it is similar to brown tumours of hyperparathyroidism and to giant cell epulis (myeloid epulis).

Clinical features. It is a painless, slowly growing tumour, expanding the jaw equally on both sides (due to presence of osteodasts). It may exhibit egg shell crackling.

Investigations

- Plain x-ray shows expanding translucent shadow with unequal trabeculae (soap bubble appearance).
- CT scan and MRI.
- Biopsy.
- Serum calcium and parathormone assay to exclude hyperparathyroidism.

Treatment

Careful enucleation and gentle curettage. In large tumours, resection of the mandible is done.

2. Osteoclastoma

This is a rare tumour that is similar to the foregoing, but is malignant on histological examination.

Malignant bone tumours

1. **Osteogenic sarcoma.** The tumour is commoner in the upper than the lower jaw. It may occur on top of Paget's disease of bones and is similar to osteogenic sarcoma anywhere.
2. **Fibrosarcoma** (parosteal fibrosarcoma). The tumour may arise from the periosteum of the jaw.
3. **Secondary carcinoma.** Metastases reach the jaw usually by direct spread from nearby carcinoma, e.g. lip, tongue, or floor of the mouth. Treatment is that of the primary together with resection of the mandible. There is no role for radiotherapy to avoid necrosis of the mandible.
4. **Malignant maxillary tumours**
 - Carcinoma
 - Columnar cell carcinoma.
 - Squamous cell carcinoma.
 - Sarcoma
 - Osteogenic sarcoma.
 - Spread from ethmoid sarcoma.
 - Malignant lymphoma (Burkitt's lymphoma).

Carcinoma of the maxilla

This is the commonest malignant tumour of the maxilla. It affects both sexes equally, usually at about the age of 40 years. Chronic infection of the antrum is predisposing factor. In most of the cases it is associated with secondary infection of the nasal sinuses.

Pathology

The tumour usually arises from the antrum and encroaches on one or more of its walls, and involves the surrounding structures. Histologically, it may be (a) Columnar cell carcinoma, if it arises from the maxillary antrum or (b) squamous cell carcinoma (less common), if it arises from the hard palate, gums or tooth sockets. The tumour spreads by infiltration of adjacent structures. Lymphatic spread occurs to the upper deep cervical and retropharyngeal lymph nodes. Blood borne metastases are rare.

Clinical features

1. In early cases, the diagnosis is suspected if pain and chronic sinusitis in an old patient fail to respond to the ordinary treatment.
2. Presentation depends on the wall of the maxillary antrum which has been infiltrated.
 - Infiltration of the anterolateral wall leads to bulging and swelling of the cheek (Fig. 22,9).
 - Infiltration of the medial wall leads to unilateral foul nasal discharge (mucous, blood and pus) due to nasal obstruction. Later on, obliteration of the nasolacrimal duct leads to epiphora.
 - Infiltration of the posterior wall encroaches on the nasopharynx and will lead to change of voice, difficulty of breathing and post-nasal discharge of pus and blood.
 - Infiltration of the roof of the antrum (floor of the orbit) will lead to proptosis and diplopia.
 - Infiltration of the floor of the antrum (the hard palate) produces depression and bulge of the palate into the mouth.
3. In late case, the patient may present with secondary lymph node enlargement.

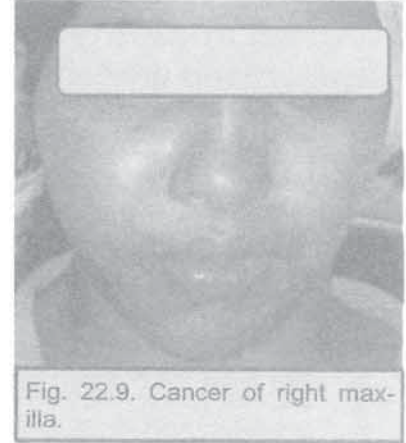


Fig. 22.9. Cancer of right maxilla.

Investigations

1. **Plain X-ray.** In early stages, the radiogram shows opacity and increase in size of the antrum. Later there is decalcification and erosion of the bone.
2. **CT scan** (Fig. 22.10) and MRI can define the exact site, size and extent of the tumour.
3. Maxillary sinus endoscopy (sinuscopy) and biopsy.

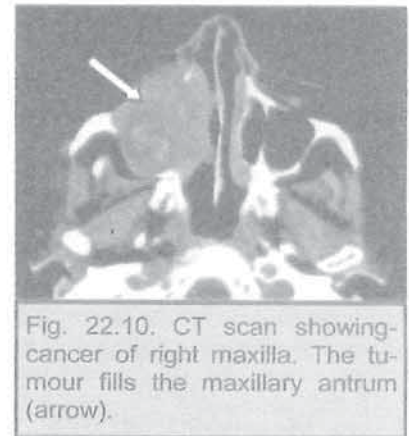


Fig. 22.10. CT scan showing cancer of right maxilla. The tumour fills the maxillary antrum (arrow).

Treatment

- Early cases. Both surgery and radiotherapy produce equal results. Surgical treatment is, however, recommended in cases with bone invasion as curability with radiotherapy is reduced in these patients.
 - The standard operation is total maxillectomy where the whole maxillary antrum is removed with its walls. A synthetic prosthesis is fitted in place of the excised bone to avoid facial deformity.
 - In the absence of lymph node involvement there is no need to perform prophylactic neck dissection. Radical block neck dissection is indicated when the cervical lymph nodes are involved by malignant deposits.
- Advanced cases. A combination of radiotherapy and surgery is better than either alone. After a course of external irradiation, maxillectomy is performed.
- Recurrent cases. Intracavitary radiation by radium needles is administered.

SALIVARY GLANDS

Surgical anatomy and physiology

Parotid gland anatomy

Position

The parotid gland lies in front of and below the lower half of the ear. It is wrapped around the vertical ramus of the mandible, with its deep portion between this part of the mandible and the mastoid process, and its superficial portion projecting forwards on the surface of the masseter. The gland reaches up to just below the zygomatic arch, and down into the neck.

Parotid fascia. The parotid is covered by a tough part of the deep cervical fascia that is distinctly named the 'parotid fascia'.

The parotid duct, also known as Stenson's duct, arises from the anterior aspect of the gland and passes forwards crossing the masseter, then dips between the fibres of the buccinator to open on the inner surface of the cheek opposite the upper second molar tooth.

The facial nerve. (Fig. 23.1) is an important relation to the gland. It comes out of the skull through the stylomastoid foramen and enters the posterior aspect of the parotid where it divides into two trunks, which give off the terminal nerve branches. These emerge from the upper, anterior, and the inferior aspects of the gland to supply the muscles of facial expression, and the platysma. Communications exist between these branches. Though there are no distinct anatomical lobes of the parotid, from the practical surgical point of view the facial nerve is described to divide the gland into a superficial and deep lobes or parts.

Other structures in the gland

- Retromandibular (posterior facial) vein.
- External carotid artery where it gives off its two terminal branches, the maxillary and the superficial temporal arteries. The facial nerve branches are the most superficial, followed by the veins, while the artery and its branches are the deepest; a fact that helps localization of the nerve during surgery.
- The parotid lymph nodes are embedded in the gland beneath the parotid fascia, while the preauricular nodes lie outside it.

CHAPTER CONTENTS

- Surgical anatomy and physiology
 - Parotid gland anatomy
 - Submandibular gland anatomy
 - Physiology
- Non-neoplastic salivary gland diseases
 - Congenital
 - Infections
 - Stones
 - Fistula
 - Degenerative
 - Autoimmune
 - Drug-induced, metabolic & endocrine
- Salivary neoplasms
 - Pathology
 - Clinical features
 - Diagnosis
 - Treatment

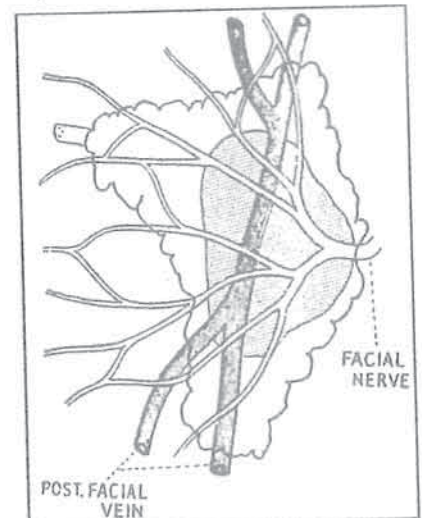


Fig. 23.1. Facial nerve and posterior facial vein in relation to parotid gland.

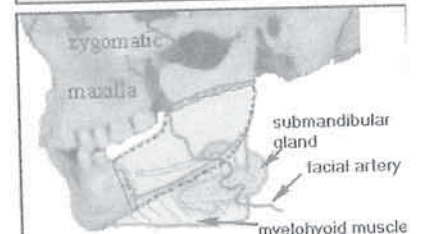


Fig. 23.2. Submandibular gland anatomy.

Nerve supply

Secretomotor fibres are originally supplied by glossopharyngeal nerve and reach the gland through the auriculotemporal branch of trigeminal nerve.

Submandibular gland anatomy

Position (Fig. 23.2). The submandibular gland is described to be formed of two parts, in relation to the mylohyoid muscle. The superficial part is present beneath the deep fascia in the digastric triangle wedged in the space between the body of the mandible and the mylohyoid. The gland hooks around the free posterior border of the muscle and extends deep to it. This small part of the gland that lies between the mylohyoid and the hyoglossus muscles is called the deep part.

Duct. From the anterior end of the deep part arises the submandibular duct. The right and left ducts open in the floor of the mouth on either side of the frenum of the tongue.

Related structures

- The lingual and hypoglossal nerves are related to the deep aspect of the submandibular gland. The lingual nerve has an intimate relation with the duct; at first the nerve is superficial to the duct, then passes below it, and finally ascends deep to the duct to supply the tongue.
- The facial artery is also closely related to the submandibular gland. At first it grooves the posterior aspect of the gland, and then passes on its lateral surface, and finally ascends over the mandible to supply the face.
- Submandibular lymph nodes lie just outside, as well as inside the gland substance. For this reason removal of the submandibular salivary gland is a necessity when radical neck lymph node dissection is being performed.

Nerve supply. Secretomotor fibres are originally supplied by chorda tympani of facial nerve and reach the gland through the lingual branch of trigeminal nerve.

Physiology

Saliva secreted by the parotid differs from that secreted by the submandibular gland. While the parotid secretes serous fluid, the submandibular gland provides larger amount of high viscosity, calcium-rich fluid. Salivary secretion is a continuous day and night process, but is heightened during eating.

Functions of the saliva

- Food lubrication to allow swallowing.
- Cleaning the mouth.
- Starch digestion by the salivary amylase.
- It mediates taste sensation.

Non-neoplastic salivary gland diseases

This term includes different disease groups which are

1. Congenital diseases; as aplasia, ectopic parotid tissue, cystic hygroma, and sialectasis.
2. Infections.
3. Salivary stones.
4. Salivary fistula.
5. Degenerative diseases.
6. Autoimmune salivary diseases.
7. Drug-induced, endocrine, and metabolic salivary gland enlargement.

Congenital Diseases

Sialectasis is the salivary gland's analogue of bronchiectasis, that is characterized by degeneration of the alveolar and duct system. As a result they become dilated. The aetiology is not known, but many cases are congenital and familial. The dilated ducts and alveoli are inadequately drained, and, thus, get infected. Affected children are usually initially diagnosed as having mumps. Unlike mumps, however, the disease is usually unilateral and recurrent. Antibiotics are used to abort the acute attacks of infection, and in general, childhood sialectasis causes no trouble after the age of 15 years.

Infections

- Acute; viral or bacterial.
- Recurrent subacute or chronic.
- Chronic infections as TB and sarcoidosis (rare).

Viral parotitis

Mumps, though diminishing in frequency because of the routine immunization, is still the commonest cause of salivary gland swelling.

Bilateral painful parotid gland swelling with fever, in a child is the usual presentation. The disease is self-limiting.

Acute bacterial sialadenitis

Bacterial parotitis is rare in modern surgical practice. In the past, it was a common occurrence in the elderly postoperative patients, because of a combination of poor oral hygiene and dehydration. Obstruction of the Stenson's duct by a stone is another cause of infection. Affection of the submandibular gland results from stone obstruction of its duct.

The commonest offending organism is *Staphylococcus aureus*. The patient complains of pain and swelling in the side of the face. Pain is marked because the swollen gland lies within the tough confines of the parotid fascia. It becomes throbbing if an abscess forms, and fever sets in. Talking exaggerate the pain.

On examination, the whole gland is found diffusely enlarged, and the skin may be red. The parotid swelling is markedly tender and feels firm. Because the gland is covered by tough fascia, the formation of an abscess does not produce fluctuation, yet is heralded by the development of skin oedema (Fig. 23.3). A stone may sometimes be felt in the duct by bimanual examination. Gentle pressure on the gland can produce a bead of pus at the duct opening.

Clindamycin is used initially, as this antibiotic attains the highest salivary concentration. Failure of 48 hour conservative treatment, or evidence of abscess formation calls for surgical drainage. The operation is done under general anaesthesia. The skin incision that is used for parotidectomy, or part of it, is employed for the abscess drainage. A skin flap is raised off the gland, and a mosquito forceps is thrust in the deep fascia at the suspected area. It is then gently opened along the direction of the



Fig. 23.3. Parotid abscess.



Evaluating a salivary gland swelling.

1. History
 - Onset, duration, course
 - Is it painful?
 - Painful diffuse swelling suggests sialadenitis
 - Painful swelling which fluctuates with meals suggests sialolithiasis
 - Non-painful swelling suggests tumour
 - Other concurrent symptoms
2. Physical exam
 - Is the mass unilateral/bilateral
 - Is it diffuse or well-circumscribed
 - Is it tender
 - Are there associated head and neck findings
 - Enlarged lymph nodes
 - Pus from duct opening
 - Palpable calculi
 - Other signs of disease
3. Investigations.
 - Plain X-rays (occlusal view) may show salivary calculi
 - Fine needle aspiration if a tumour is suspected
 - CT or MRI if a tumour or abscess is suspected

fascial nerve branches to avoid injuring them (Hilton's method). Pus is sampled for culture and sensitivity. A rubber drain is inserted, and is kept for a few days till the discharge stops.

Reurrent subacute and chronic sialadenitis

These conditions are usually consequent upon an abnormality in the salivary gland, e.g. sialiectasis, stones, or autoimmune diseases.

Chronic sialadenitis of the submandibular salivary gland presents as a swelling in the digastric triangle which may be confused with submandibular lymph nodes. The disease is characterized by the following

- History of pain and increase in the size of the swelling during eating.
- The swelling is solitary and cannot be rolled over the edge of the mandible.
- Inspection of the floor of the mouth may reveal redness of the orifice of the duct.
- Bimanual palpation reveals that the swelling is filling the floor of the mouth.

Salivary stones (sialolithiasis)

Pathology. Stones sometimes form from the constituents of saliva. They may be the result or cause of chronic and recurrent sialadenitis. They may also complicate cases of Sjogren's syndrome because of the reduced flow of saliva. As the saliva of the submandibular gland is viscous and has the highest calcium concentration, the incidence of submandibular stone formation is much higher than the parotid and the sublingual glands.

The stone either resides in the gland or in the duct. Salivary mud or stones can block the minor or major ducts, causing either localized or generalized painful gland swelling while eating. A calculus may also cause acute or recurrent attacks of sialadenitis (Fig. 23.4)



Fig. 23.4. Submandibular sialadenitis caused by a stone.

Investigations. Because of its high calcium content, a stone in the submandibular gland is radio-opaque in 80% of cases and can be seen on a plain occlusal view radiograph (Fig. 23.5). In contrast, most of parotid stones are radiolucent. They show as filling defects or blocks to sialography.

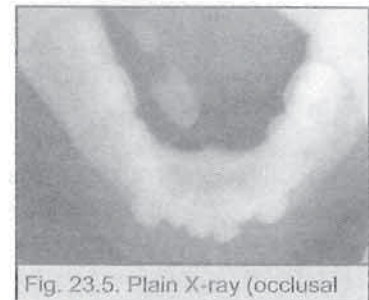


Fig. 23.5. Plain X-ray (occlusal)

Treatment of stones is surgical. A stone that is palpable within a duct can be removed through the mouth, but a stone within the gland substance must be removed with the whole submandibular or parotid gland. Parotidectomy for calculi disease demands high experience as the recurrent inflammation makes identification of the facial nerve very difficult.

Salivary fistula

A fistula that discharges saliva to the skin of the cheek may result from accidental trauma injuring the parotid gland or duct. Less commonly it complicates surgery on the gland. In some rare cases an internal fistula results from ulceration of a duct stone in the mouth cavity, which is a harmless condition that deserves no treatment.

A fistula arising from the gland substance usually heals spontaneously, but one that arises from the duct is unlikely to heal on its own because of the high rate of salivary flow. In the latter a sialogram is needed to exclude distal duct obstruction by stone or

stricture. Surgical treatment is usually necessary. The distal part of the duct is cannulated from the mouth and a trial is made to repair it without tension. If this fails, the gland is excised.

Degenerative Diseases

Sialectasis

Sialectasis means abnormal dilatation of the small branches of the salivary ducts and the salivary alveoli. The cause of this degenerative disease is not known, but as mentioned before, the childhood type is known to be a familial disease. Punctate sialectasis is also sometimes seen on sialography for cases of Sjogren's syndrome. The degenerating alveoli coalesce and become cystic. The ducts become strictured and in places dilate.

The usual presentation is by recurrent attacks of sialadenitis. Sialography is the investigation of choice for suspected sialectasis. The salivary duct is cannulated and 0.5-2 ml of a radio-opaque medium, as Lipiodol or Hypaque (sodium diatrizoate), are instilled into the duct. Films are taken before and after the patient sucks a lemon for several minutes (postevacuation film). The stenosed and dilated duct and branches are visualized, and the cystic coalition of alveoli shows as a snow storm appearance" (Fig. 23.6).



Fig. 23.6. Sialography.
•Normal parotid sialogram.
•Parotid sialectasis.

Initial treatment of sialectasis is conservative. The patient is advised to finish every meal by a citrus drink to stimulate salivary flow, and then to massage the affected gland to milk out the accumulating epithelial debris. Antibiotics, e.g., clindamycin, are used to treat attacks of infection. Surgical excision of the gland is indicated in troublesome cases.

Radiation sialadenitis

This may be caused by radiation to the nasopharynx or the skull base. Salivary secretion is temporarily suppressed, and the patient can be helped by the administration of sialogogues as citrus fruits.

Autoimmune salivary diseases

Types

1. **Sjogren's disease (pronounced shogren's).** The disease is by far commoner in women than men. Its manifestations include dryness of the mouth (xerostomia), dryness of the eye (xerophthalmia, keratoconjunctivitis sicca), as well as rheumatoid arthritis. Some patients complain of salivary gland discomfort or enlargement (sialomegaly). The aetiology is not exactly known, but the disease is thought to be caused by a cytomegalovirus which affects the ducts of the salivary glands, rendering them antigenic. The result is infiltration of the glands with lymphocytes around the ducts, obliterating them. Patients with Sjogren's disease are 44 times more prone to the development of lymphoma than the general population. This is attributed to an alteration in the relationship between the T-helper and the suppressor cells.
2. **Benign lymphoepithelial lesions.** This is an uncommon disease that is characterized by progressive lymphocytic infiltration and diffuse enlargement of

salivary glands particularly the submandibular and the parotid. The disease most commonly affects middle aged and elderly women. The term benign is a misleading because of its prelymphomatous potential.

Complications

20% of cases develop lymphomas.

Diagnosis

- Lip biopsy that includes the minor salivary glands.
- Parotid sialography shows the nonspecific appearance of narrowed ducts and sometime punctate sialectasis.

Treatment

- Instillation of artificial tears that are made up of methylcellulose helps to combat eye dryness.
- Meticulous oral hygiene.
- If a patient with these autoimmune salivary diseases develops a palpable mass in the parotid, needle biopsy should be performed. If this proves to be a lymphoma, the gland is removed, the disease staged, and treatment is continued with radio and/or chemotherapy.

Drug-induced, metabolic and endocrine salivary gland enlargement

Drug-induced enlargement of the salivary glands has been reported with sulfisoxazole, phenylbutazone, iodide containing compounds, thiouracils, hypotensive drugs, and contraceptive pills. Metabolic and endocrine causes include liver cirrhosis, diabetes, alcoholism, malnutrition, and ovarian, pancreatic, or thyroid insufficiency.

Salivary neoplasms

Salivary neoplasms constitute 1.2% of all neoplasms and 5% of head and neck tumours.

Pathology

The majority of these neoplasms are benign and most commonly arise in the parotid gland. The incidence of malignancy varies inversely with the size of the gland, thus it occurs in 25% of parotid neoplasms, in 40% of submandibular neoplasms, and in 70% of neoplasms of the sublingual and minor salivary glands.

These tumours can arise from the secretory tissue, the duct system, or from the lymphoid tissue. There are many types and different classifications.

Benign neoplasms

- By far the commonest salivary neoplasm is the **pleomorphic adenoma** of the parotid. This was referred to in the past as mixed salivary tumour because of its histological appearance. Epithelial cells arranged in sheets and duct-like structures are interspersed by mucoid material, which was thought to be cartilaginous. This tumour is characterized by an incomplete capsule that allows extension of the neoplastic epithelium into the surrounding tissues. It is slowly growing without infiltrating the facial nerve. Long-standing (more than 10 years) pleomorphic adenomas rarely turn into carcinoma.
- An **adenolymphoma** is usually found in the lower part of the parotid gland and is bilateral in 10% of cases. It is thought to arise from heterotopic salivary tissue in the parotid lymph nodes. Microscopically, the tumour is formed of epithelial lined spaces

filled with creamy material, that are surrounded by lymphoid tissue. It tends to affect elderly people and is related to smoking.

- **Haemangiomas and lymphangiomas** may affect the parotid, but these are pathologically hamartomas rather than true neoplasms.

Malignant neoplasms

- Mucoepidermoid carcinoma arises from the duct epithelium and is the commonest member of the group. It usually affects the parotid. Three grades are described; low, intermediate, or high-grade tumours. The low-grade type is the most frequent and is known to affect children.
- Adenoid cystic carcinoma is the commonest malignancy affecting the minor salivary glands. Its distinctive features are the slow rate of growth, and its perineural spread, i.e., it infiltrates for a long distance in the perineural tissues of adjacent nerves. For this reason the tumour is likely to be incompletely removed, and hence it has a high recurrence rate.

Clinical features

Benign neoplasms

- The patient complains of a painless swelling on the side of the face which has been present for months or years and which is slowly growing, or has stopped growing.
- On examination, there is a localized swelling, usually in the parotid, which is hemispherical and may attain a large size (Fig. 23.7). The mass is nontender, firm in consistency (an adenolymphoma feels soft or cystic), and has a smooth or bosselated surface. It does not infiltrate the skin, the masseter nor the mandible.
- An important point is that there is no facial nerve affection.
- The cervical lymph nodes are not enlarged, and there is no evidence of distant metastases.
- Tumours arising in the deep part of the parotid may bulge in the oropharynx behind the tonsil, hence the importance of examining the mouth cavity.

Malignant neoplasms

- The patient commonly complains of a steadily enlarging swelling on the side of the face. Salivary malignancies do not usually grow as fast as other cancers in the body. The swelling is sometimes painful, and pain may radiate to the ear, and is intensified by mastication.

COMMON SALIVARY NEOPLASMS

Benign

- Pleomorphic adenoma
- Adenolymphoma (Warthin's tumour)
- Oncocytoma (oxyphil adenoma)
- Monomorphic adenoma

Malignant

- Mucoepidermoid carcinoma
- Adenoid cystic carcinoma
- Acinic cell carcinoma
- Adenocarcinoma
- Carcinoma ex pleomorphic adenoma

The commonest salivary neoplasm is pleomorphic adenoma of the parotid.

Parotid pleomorphic adenoma is important for three reasons.

1. It is very near to the facial nerve which can be damaged at operation.
2. If the capsule is damaged in removal or if it was just enucleated, recurrent disease is possible.
3. It can turn malignant, and when it does it is dangerous.

Warthin's tumour is

- Soft and cystic.
- Usually affects older people.
- Not dangerous and only needs to be operated on for cosmetic reasons or if diagnosis is unsure.

Malignant parotid tumours should be suspected with

- Short duration of growth.
- Pain or tenderness,
- Classical signs of facial nerve palsy or metastatic cervical lymph nodes are uncommon unless the disease is locally advanced.

- The patient may give a history of a painless swelling that has been stationary for years and is now getting bigger (carcinoma ex pleomorphic adenoma).
- The mass is usually warm and mildly tender. It is firm or hard and has an irregular surface. It may be adherent either to the skin, the masseter or the mandible.
- If the tumour infiltrates the facial nerve, there is evident weakness or paralysis of the facial muscles (Fig. 23.8).
- The cervical lymph nodes are sometimes involved. Rarely, these tumours metastasize to the lungs.



Fig. 23.7. Pleomorphic adenoma of parotid. It typically raises the lobule of ear and does not affect facial nerve.

Differential Diagnosis

1. Extra parotid swellings
 - a. Lymph nodes (parotid or upper deep cervical), sebaceous cysts, and lipomas may mimic pleomorphic adenomas.
 - b. Mandibular and maxillary tumours may produce the appearance of a parotid enlargement.
 - c. Hypertrophy of the masseter is bilateral in most cases. It is usually seen in ladies who have the habit of involuntary grinding of their teeth, or in those who have had orthodontic treatment. The condition is sometimes so difficult to differentiate from a true parotid enlargement, that a CT scan is sometimes resorted to for diagnosis.
2. True parotid enlargement that is caused by the already mentioned non-neoplastic salivary gland diseases. The gland is diffusely enlarged with no definite lump.



Fig. 23.8. Malignant parotid tumour with facial paralysis.

Investigations

In most cases of salivary gland lumps, clinical diagnosis is reliable enough to proceed to treatment. In a minority of cases, however, ancillary investigations are needed.

Pathological diagnosis

In general, a pathological diagnosis is not a necessity for tumours that behave in a benign way, but is essential before operating on tumours that are clinically malignant. Surgery for the latter is extensive, and sometimes mutilating, as it may entail sacrifice of the facial nerve, and one should be backed with a pathological proof of malignancy before operation. Until recently, biopsy of the parotid gland was feared because of the risk of spillage of cells, even from pleomorphic adenomas, leading to recurrence. It is also liable to result in injury of the facial nerve, or the formation of a salivary fistula. The development of fine needle aspiration cytology (FNAC) obviates these risks, but is reliable only in the hands of an expert cytologist. For tumours of the minor salivary glands of the mouth cavity, excision biopsy is feasible.

Open surgical biopsy of the major salivary glands is contraindicated. FNAC is safer.

Imaging

CT scan (Fig. 23.9) and MRI are the most useful methods for assessment, particularly for tumours arising from the deep part of the parotid.

Isotope scan. In general, a salivary neoplasm shows as a cold spot on isotope scanning with technetium (Tc^{99m}) pertechnetate. Adenolymphoma and oncocytoma are exceptions as they concentrate the technetium, and thus show as hot spots.

Treatment

Surgery is the only reliable form of treatment of salivary neoplasms. The operation should be carried out by a surgeon with experience in this field. For parotid tumours, the patient should be warned against the possibility of accidental facial nerve injury, or intentional sacrifice if the lesion is malignant. Even when the nerve is successfully preserved, patients commonly develop nerve weakness because of neurapraxia that recovers spontaneously a few months after the operation.

Tumours that are clinically benign

Enucleation of a pleomorphic adenoma is easy, but is followed by recurrence that is caused by the left-over tumour extensions through the defective capsule. The tumour should, therefore, be excised with a safety margin.

As the majority of the tumours arise in the parotid part that is superficial to the facial nerve, the standard operation is superficial parotidectomy (Fig. 23.10). Early in the course of the operation the facial nerve trunk is exposed. All the parotid tissue that is superficial to the nerve and its branches is excised, taking great care not to injure them. Tumours arising from the deep part of the gland are treated by total conservative parotidectomy, i.e. excising the whole gland while preserving the facial nerve integrity. In case of accidental nerve injury immediate repair is performed by microsurgical techniques either by direct suturing, or by a nerve graft taken from the great auricular nerve.

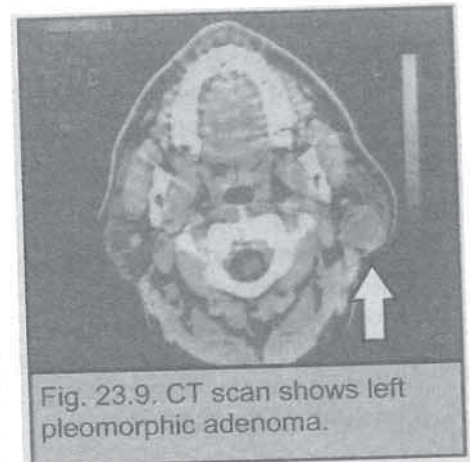


Fig. 23.9. CT scan shows left pleomorphic adenoma.

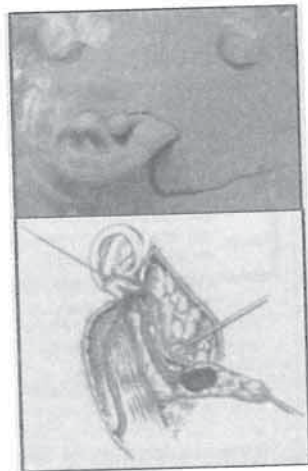


Fig. 23.10. Superficial parotidectomy. The commonest indication is a pleomorphic adenoma.
1. Incision.
2. Removal of the parotid tissue superficial to facial nerve and branches. This is usually the diseased part.
3. Facial nerve preserved.

Benign tumours of the submandibular gland are treated by the operation of submandibular sialadenectomy (Fig. 23.11). An incision is made parallel to and 2 cm from the lower border of the mandible to ensure safety of the mandibular division of facial nerve. Two other nerves are at risk when dissecting the deep aspect of the gland; which are the

lingual and the hypoglossal nerves. These should be exposed and preserved. Likewise tumours of the minor salivary glands are treated by simple excision with a safety margin.

Tumours that are clinically malignant

If a pathological diagnosis has not been obtained prior to the operation, frozen section examination during surgery is helpful. The standard treatment is radical excision which entails wide surgical clearance with cervical lymph node dissection if they were enlarged. For the parotid, this usually necessitates excision of the facial nerve, and probably part of the masseter or the mandible.

Treatment is modified according to the aggressiveness of the tumour. For the low-grade mucoepidermoid carcinoma, an attempt to preserve the facial nerve is warranted. On the other hand, for the high-grade variety, surgery should be more extensive, and radical node dissection should be performed even if the lymph nodes were not enlarged.

Radiotherapy is of limited value, but is administered as a postoperative adjuvant therapy for tumours of high grade malignancy.

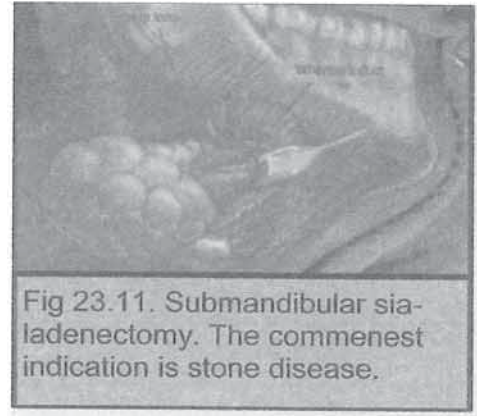


Fig 23.11. Submandibular sia-
ladenectomy. The commonest
indication is stone disease.

THE NECK

Anatomical divisions of the neck

Triangles

Each side of the neck is divided by the sternomastoid muscle into two main triangles (Fig. 24.1).

Posterior triangle.

Anterior triangle, which is further divided into

- Submandibular triangle, which is bound by body of the mandible above, posterior belly of digastric posteriorly and anterior belly of digastric anteriorly.
- Carotid triangle, which is bound by the sternomastoid muscle posteriorly, the posterior belly of the digastric superiorly and, the superior belly of omohyoid inferiorly and anteriorly.
- Muscular triangle, which is bound by the sternomastoid muscle posteriorly, the superior belly of omohyoid superiorly and the middle line anteriorly. The two muscular triangles form together a diamond shaped area.

CHAPTER CONTENTS

- Anatomical divisions of the neck
- Congenital anomalies
- Neck injuries
- Cellulitis of the neck
- Cervical lymphadenopathy
- Differential diagnosis of neck swellings
- Thoracic outlet syndrome
- Radical block neck dissection

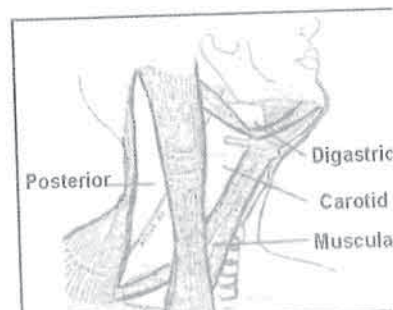


Fig. 24.1. Triangles of the neck.

Compartments

In a cut section the neck is divided into two compartments; the musculo-skeletal and the visceral compartments (Fig. 24.2).

- The muscular compartment lies posteriorly and is separated from the visceral compartment by thick fascia; the prevertebral fascia. In addition, the visceral compartment is surrounded by the deep fascia (investing fascia) of the neck.
- The visceral compartment lies anteriorly. It contains all viscera and most of the vessels and nerves of the neck, e.g., oesophagus, trachea, thyroid and parathyroid glands, carotid arteries, vagus nerve and the jugular veins. Because the visceral compartment is enclosed within such a fascial covering any swelling within the visceral compartment can cause symptoms and signs due to pressure on its different viscera, e.g., dysphagia, dyspnoea or weak carotid pulse.

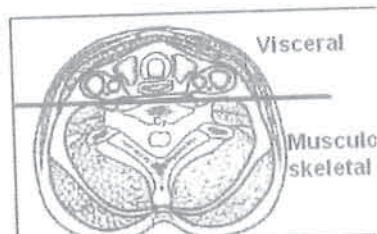


Fig. 24.2. Compartments of the neck.

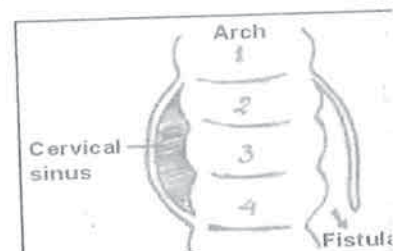


Fig. 24.3. Development of branchial cyst and fistula.

Congenital anomalies

Branchial cyst and fistula

Aetiology

Normal development

During embryological development the second branchial arch grows rapidly covering the 3rd and the 4th arches then it fuses with the neck. The space between the second arch and the rest of the arches turns into a cervical sinus which soon disappears.

Abnormal development (Fig. 24.3)

- If the cervical sinus persists it becomes a branchial cyst.
- A branchial fistula develops if the second arch doesn't completely fuse with the neck.

Pathology

- A **branchial cyst** is lined by squamous epithelium and usually contains a clear fluid which is rich in cholesterol crystals that can be microscopically identified in aspirated cyst fluid (Fig. 24.4). The cyst wall is surrounded by lymphatic tissue thus the cyst becomes susceptible to infection.
- A **branchial fistula** is usually congenital but occasionally develops secondary to ruptured infected branchial cyst. The track is lined by squamous or ciliated epithelium and extends up to the side wall of the nasopharynx (fossa of Rosenmuller). The track in its course passes between the bifurcation of the common carotid artery, deep to the posterior belly of digastric muscle and superficial to the hypoglossal nerve.

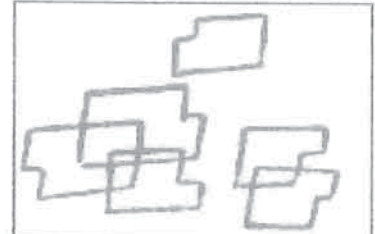


Fig. 24.4. Cholesterol crystals are rectangular with one missing angle.

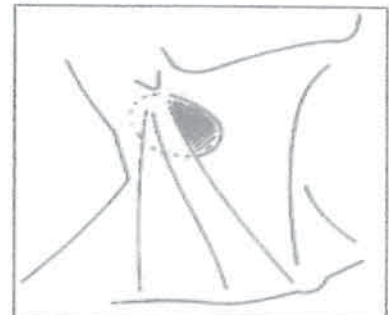


Fig. 24.5. Site of branchial cyst.

Clinical features

Branchial cyst

- The cyst usually appears at late childhood.
- It presents as a swelling which protrudes from beneath the anterior border of the upper third of the sternomastoid muscle (Fig. 24.5).
- The swelling has a smooth surface, is mobile and fluctuant.
- A branchial cyst is commonly mistaken for a cold abscess but can be differentiated from it by finding cholesterol crystals in the aspirate.

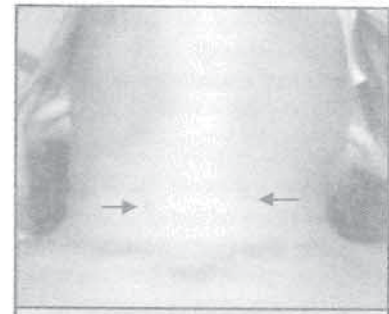


Fig. 24.6. Bilateral branchial fistulae present as tiny openings.

Branchial fistula

- A branchial fistula presents at birth as a pin point opening at the anterior border of the lower third of the sternomastoid muscle (Fig. 24.6).
- The opening discharges mucoid material but when the track is infected the discharge becomes purulent.
- A branchial fistula may occasionally be confused with a tuberculous sinus.

Treatment

Branchial cyst. Treatment is excision through a transverse neck incision.

Branchial fistula. The whole track should be excised. This is done through multiple transverse neck incisions. A small one around the fistula opening, and the other at a higher level below the jaw. A ureteric catheter is introduced into the track to facilitate its identification during surgery.

Thyroglossal cyst and fistula (Chapter 26)

Cystic hygroma

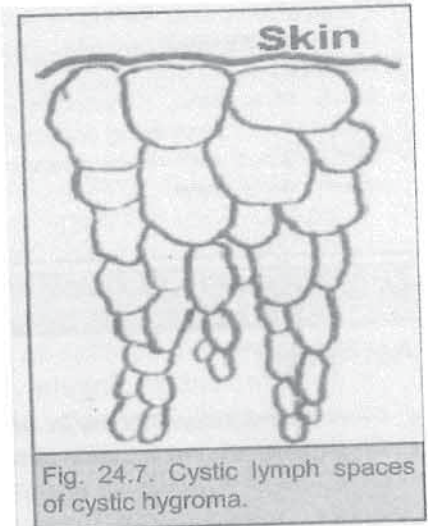
Aetiology

Normal development The lymphatic system develops by the coalescence of multiple small lymph vesicles. A large accumulation of these lymph vesicles is present lateral to the jugular vein and is called the jugular lymph sacs.

Abnormal development If some of the vesicles of the jugular lymph sac fail to join the lymphatic system they become sequestered and form a cystic hygroma. The most common site is in the neck. However, a similar lesion can arise in the cheek, tongue, axilla, mediastinum or in the groin.

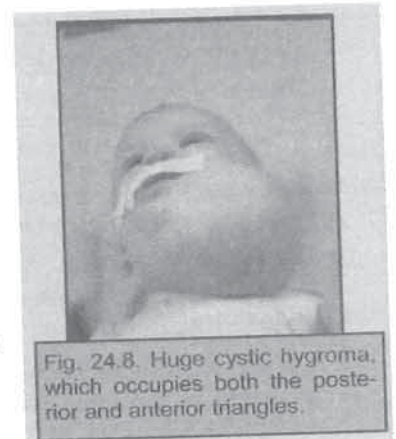
Pathology

- The swelling consists of multiple cysts with the larger ones near the surface while the smaller ones are deep (Fig. 24.7) and infiltrating tissue planes.
- Each cyst is lined by endothelial cells and contains clear lymph.



Clinical features

- The condition usually presents at birth or within the first few years of life.
- It presents as a painless swelling that is usually situated in the lower part of the posterior triangle (Fig. 24.8).
- The swelling appears bluish as the overlying skin is thin and the swelling is translucent to light.
- The swelling is soft and is partially compressible and increase in size with coughing and crying.
- The swelling may occasionally increase rapidly in size to the extent that it may interfere with respiration.



Treatment

Excision of the swelling at about the age of 3-years should be done. This could be facilitated by preoperative injection of boiling water in the swelling to induce fibrosis to make it smaller.

Sternomastoid tumour and congenital torticollis

Aetiology

- **Normal development** The sternomastid muscle develops through the union of three somites; each with its own blood supply.
- **Abnormal development** Interruption of the blood supply to the central somite of sternomastoid muscle occurs either before or at birth. This causes muscle infarction. The infarcted part becomes swollen, hence the name sternomastoid tumour. After while the infarcted part will be replaced by fibrous tissue that will contract causing congenital torticollis.

Clinical features

- At birth (or shortly afterwards) there is a painless firm swelling at the middle of the sternomastoid muscle.
- Later when torticollis develops the head will be tilted to the side of the lesion with the face looking to the opposite side.
- If the condition is neglected it leads to facial asymmetry.

Differential diagnosis

This condition should be differentiated from 'wry neck' which is fibrositis causing spasm of the sternomastoid muscle. This condition is temporary lasting for a day or two and responds to anti-inflammatory drugs.

Treatment

- Early after birth. Attempt to prevent the development of the deformity by stretching and splinting of the neck may be done.
- Established deformity. Division of the sternomastoid at its lower part is done.



Fig. 24.9. Sternomastoid tumour and congenital torticollis.

Neck injuries

Neck injuries may be penetrating or non-penetrating. Penetrating injuries are much more common. They may be caused by gunshots or stabs. Blunt injuries usually occur secondary to road traffic accidents and may cause fracture of the spine.

The most commonly injured structures are the big vessels, spinal cord and the aerodigestive tract in that order.

Neck injuries are serious because:

1. The neck contains vital viscera and vessels,
2. Unstable cervical spine fracture and dislocations can cause paraplegia, quadriplegia, or immediate death.
3. Neck injuries are commonly associated with head, face, and chest injuries.

Vascular injuries Present by

- Hemorrhage which may be severe enough to cause shock.
- Expanding hematoma.
- Injury to a large vein may lead to pulmonary embolism.

Neurological injuries

- Fracture of the cervical spine may lead to quadriplegia or paraplegia.
- Injury of the brachial plexus or cranial nerves.

Injuries of the larynx or trachea may present by respiratory obstruction, aphonia, hoarseness of voice, subcutaneous emphysema, air bubbling through the wound or hemoptysis.

Esophageal injuries will be followed by leakage and severe infection in the neck which may spread to the mediastinum.

Investigations

1. Plain X-ray may reveal foreign bodies, wide mediastinum, subcutaneous emphysema or fractures.
2. Duplex ultrasound may reveal injury to the big vessels.
3. CT of the skull and brain.
4. CT angiography.
5. Laryngoscopy and bronchoscopy for laryngotracheal injuries.
6. Gastrografin swallow for esophageal injuries.

Treatment

1. **ABCDE** of trauma must be followed
 - a. Airway management especially endotracheal intubation can be both difficult and dangerous. It must be done by the most experienced personnel.
 - b. Cricothyroidotomy may be easier and safer than tracheostomy.
 - c. Trendelenburgh position helps to avoid air embolism in major venous injuries.
 - d. I.V. lines should be avoided in the side of trauma.
2. **Tension pneumothorax** is managed by inserting a needle in the second intercostal space.
3. **Minor external bleeding** can be managed by local compression. Major bleeding needs exploration to stop the bleeding and perform vascular repair.
4. In case of **neurologic damage**, neck support and evaluation of the patient initially according to the Glasgow coma scale, then by CT and/or MRI to define the exact neurological lesion.
5. **Oesophageal** injury if discovered early is treated by immediate exploration and repair
6. **Laryngotracheal** injuries need surgical repair.

Cellulitis of the neck

Cellulitis of the neck is either superficial or deep to its deep fascia.

Superficial cellulites

Superficial cellulitis is common and is diagnosed and treated as cellulitis anywhere else. Rarely superficial cellulitis above the level of the hyoid bone can lead to sudden asphyxia especially in children from sudden oedema of the glottis.

Deep cellulites (Ludwig's angina)

Pathology

Ludwig angina is a clinical condition that is characterized by inflammatory swelling of both the submandibular region and the mouth cavity. It is due to deep cellulitis around the submandibular gland usually due to streptococcal infection. Infection is deep to the deep fascia and can spread to the glottis causing oedema and asphyxia.

Clinical features

- The patient presents with an inflammatory swelling in the submandibular region. The swelling is painful, red, warm, tender and the overlying skin is oedematous (Fig. 24.10).
- Swelling of the tongue which is pushed upwards and forwards.



Fig. 24.10. Ludwig's angina.

Treatment

Antibiotics and drainage through an incision beneath the jaw which is deepened to permit displacement of the submandibular salivary gland and division of the mylohyoid muscle to allow for adequate drainage.

Cervical lymphadenopathy

Half of the lymph nodes of the body are present in the neck. The main cervical lymph node groups are shown on Fig. 24.11. These nodes drain the head and neck. In addition, the supraclavicular nodes are secondary stations for the breast, apex of lung, upper limbs, as well as abdominal viscera and testes.

Accordingly cervical lymphadenopathy is very common. The causes of lymph node enlargement (lymphadenopathy) are discussed in chapter 15.

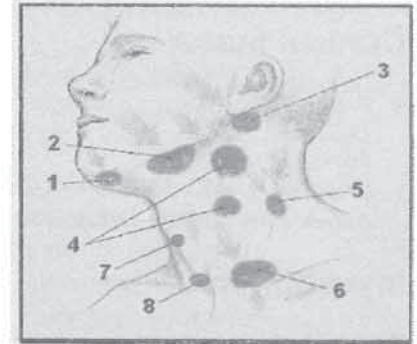


Fig. 24.11. Cervical lymph nodes

1. Submental.
2. Submandibular.
3. Jugulo-digastric (part of upper deep cervical).
4. Upper and mid deep cervical.
5. Posterior triangle.
6. Supraclavicular (part of lower deep cervical). They drain
 - Other post triangle nodes
 - Thyroid
 - Pharynx
 - Larynx
 - Upper oesophagus
 - Breast
 - Upper limb
 - May be involved in drainage of apical lung cancer (Pancoast tumour)
 - On left side may be involved in drainage of malignancies below the diaphragm, e.g., gastric, pancreatic and testicular cancer.
7. Prelaryngeal.
8. Pretracheal.

Differential diagnosis of neck swellings

The diagnosis of a mass in the neck depends upon:

- Age of the patient.
- Clinical course.
- Solid or cystic swelling.
- Site.

The most common neck swellings are

1. Enlarged lymph nodes. These are recognized by being at their known anatomical sites, and by their multiplicity.
2. Thyroid swellings. These are recognized by being in the anatomical site of thyroid gland (muscular triangle), and by their movement with deglutition.

Mid-line swellings

Solid swellings

1. Submental lymph node enlargement.
2. Nodule in the isthmus of the thyroid gland.

Cystic swellings

1. Dermoid cyst.
2. Thyroglossal cyst.(see thyroid gland).
3. Subhyoid bursitis. This is a rare tender, oval swelling, which lies transversely below the hyoid bone. It moves up and down with deglutition and with protrusion of the tongue.
4. Laryngocoele. This swelling occurs in musicians playing with air-blown instruments. It is actually a herniation of laryngeal mucosa through the thyrohyoid membrane. The swelling is resonant, compressible and increases in size with coughing or blowing.
5. Cysts in the thyroid gland
6. Cold abscess, which is rare in the mid-line.

Swellings in the submandibular triangle

1. Enlarged submandibular lymph nodes.
2. Enlarged submandibular salivary gland.

They could be differentiated from each other as the lymph nodes are multiple, could be rolled over the edge of the mandible and, unlike submandibular salivary gland swellings, can not be felt in the floor of the mouth.

Swellings in the carotid triangle**Solid swellings**

1. Enlarged upper deep cervical lymph nodes.
2. The upper part of an enlarged lateral lobe of the thyroid gland.
3. Carotid body tumour. This is a rare slowly growing malignant tumour arising from the chemoreceptors present at the bifurcation of the carotid artery. It usually presents at middle age as a slowly growing swelling which is usually smooth but may be lobular. The swelling moves from side to side but not vertically. It exhibits transmitted pulsations from the underlying carotid artery. Angiography can prove the diagnosis. Treatment of this tumour is excision with preservation of the internal carotid artery or if its preservation is not possible the artery should be replaced by a graft even before excision to avoid interrupting the blood flow to the brain.

Cystic swellings

1. Cold abscess.
2. Branchial cyst.

Swellings in the posterior triangle**Solid swellings**

1. Enlarged lymph nodes.
2. Neurofibroma arising from the brachial plexus.
3. Cervical rib.

Cystic swellings

1. Cystic hygroma.
2. Pharyngeal pouch (chapter 25).
3. Cold abscess.
4. Pneumatocoele. This is a cystic swelling in the supraclavicular region, which is resonant and compressible. It is due to herniation of the pleura into the base of the neck.

Other swellings that may arise anywhere

In addition, swellings of the skin and subcutaneous tissue are common in the neck region and should be put in mind. They are added to any of the previous lists.

1. Lipomas.
2. Sebaceous cysts.
3. Haemangiomas.

Thoracic outlet syndrome

The brachial plexus and the subclavian artery pass to the upper limb through a narrow triangle in the base of the neck (Fig. 24.12). This triangle is made of the scalenus anterior muscle anteriorly, the scalenus medius muscle posteriorly and the first rib inferiorly. At this narrow space compression of the nerves and the artery may occur causing symptoms and the development of the thoracic outlet syndrome.

Aetiology

Causes of such a compression may be

1. Cervical rib which may be complete or incomplete. This bony structure extends from the 7th cervical vertebra to the first rib.
2. A fibrous band extending from an incomplete cervical rib which ends at the first rib.
3. A tight scalene muscle.
4. Post fixation of the brachial plexus. In this case the lower root of the brachial plexus arises from T₂ instead of T₁ thus this nerve becomes excessively bend over the first rib.

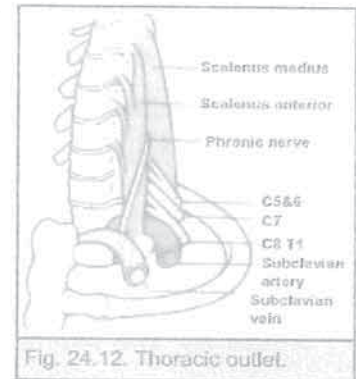


Fig. 24.12. Thoracic outlet.

Pathology

- **Brachial plexus:** The lower trunk of the brachial plexus is the main structure to be compressed. It contains sensory fibers to medial aspect of the hand and forearm and motor fibres to small muscles of the hand. In addition, it contains most of the sympathetic fibers of the upper limb.
- **Subclavian artery** Less commonly the subclavian artery is compressed causing
 - Chronic ischaemia of the upper limb.
 - Post-stenotic dilatation in the segment immediately following the compression (Fig. 24.12).
 - Post-stenotic dilatation may develop a thrombus inside. This thrombus may send a shower of emboli to the index and middle fingers as they are the direct continuation of the brachial artery. These emboli can produce digital gangrene.
- **Subclavian vein** Compression of the vein produces
 - Subclavian and axillary vein thrombosis.
 - Chronic venous insufficiency of the upper limb

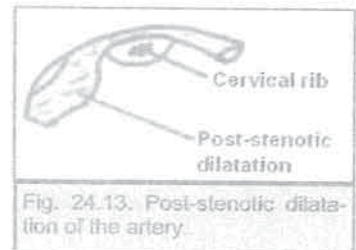


Fig. 24.13. Post-stenotic dilatation of the artery.

Clinical features

The condition occurs more frequently in females. The symptoms rarely develop before adulthood. It is usually unilateral affecting the right arm (dominant arm), but is sometimes bilateral.



Fig. 24.14. Plain X-ray shows a bony cervical rib on the left side and a large transverse process of C7 on the right side.

Symptoms

The patient may complain of one or more of the following:

- **Nerve compression symptoms** Tingling and numbness in the medial aspect of the forearm and the hand due to compression on the lower root of the brachial plexus
- **Arterial symptoms**
 - Intermittent claudication pain in the upper limb which appears with exercise.
 - Raynaud's phenomenon which occurs in cold weather. The patient will complain of attacks of pallor in the fingers followed by cyanosis, and then

A cervical rib can be present without symptoms. Its accidental discovery on radiography deserves no treatment.

redness (rebound hyperaemia). This phenomena occurs due to irritation of the sympathetic fibres supplying the upper limb.

- **Venous symptoms are rare**
 - Acute upper limb pain and swelling (DVT).
 - Chronic heaviness and swelling (chronic venous insufficiency).

Signs

- Examination rarely reveals a bony swelling at the base of the posterior triangle, which is the bony cervical rib.
- Hyposthesia may be detected in the medial forearm and the hand. Mild wasting of hand muscles may be noticed.
- A weaker radial pulse than the opposite side may be detected.
- Adson's test may demonstrate compression of the subclavian artery. In this test the patient is asked to extend his head, look at the opposite side and take a deep breath. The examiner then palpates the radial pulse while the arm is pulled down. If the test is positive the radial pulse strength is reduced. However, it should be noted that this test is not very accurate.

Differential diagnosis

- Other causes of localized pressure on the nerve
 - Cervical spondylosis
 - Carpal tunnel syndrome.
- Other causes of Raynaud's phenomenon
 - Raynaud's disease.
 - Systemic lupus and other collagen disease as they may be associated with vasculitis.

Investigations

1. Plain X-ray to the neck may detect a bony cervical rib (Fig. 24.).
2. Nerve conduction study between the neck and the forearm to detect delay in conduction. This is a useful investigation and can differentiate between thoracic outlet syndrome and carpal tunnel syndrome
3. Arterial and venous duplex ultrasound if there are vascular problems.

Treatment

Early cases

Physiotherapy to strengthen shoulder muscles may relieve the symptoms.

Failure of conservative treatment

Surgery is done to relieve the compression. This can be achieved through any of the following operations

1. Excision of a bony cervical rib.
2. Division of the scalenus anterior muscle (scaleneotomy) to relieve the compression anteriorly.
3. Excision of the first rib to relieve the compression inferiorly. This is a very efficient operation and can be done through a trans-axillary approach.
4. Endovascular stenting of the stenosed segment of the subclavian artery.

Radical block neck dissection

Definition

Removal of all lymph nodes on one side of the neck, in one mass (en bloc).

Aim

The aim is eradication of malignancy, aiming at cure. This is achieved by removing the primary lesion and its lymph node metastases by block dissection.

Indications

1. Operable primary malignancy of the head or neck, with palpable malignant cervical lymph nodes. This is the main indication.
2. Prophylactic radical block neck dissection, i.e., where cervical nodes are impalpable, is done in malignancies that are known for their high tendency to lymphatic spread. These include melanoma and carcinoma of the tongue. In other types of malignancies if the lymph nodes are not palpable the primary is removed then the patient should be followed up every 2 months. If the lymph nodes become enlarged block neck dissection is done.

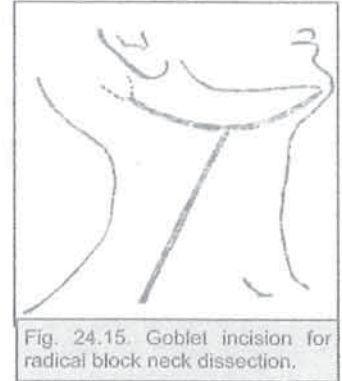


Fig. 24.15. Goblet incision for radical block neck dissection.

Technical considerations

The operation can be done through different incisions. The goblet incision (Fig. 24.15) is a popular one.

Structures that are removed in radical block neck dissection

1. All lymph nodes on one side of the neck.
2. Other structures are also removed because of their close proximity to the cervical nodes.
 - The sternomastoid muscle.
 - The carotid sheath.
 - The internal jugular vein.
 - The submandibular salivary gland.
 - The lower part of the parotid gland.
 All these structures are removed as one mass (Fig. 21.11).

Structures to be preserved in radical block dissection

1. The carotid artery.
2. The vagus nerve.
3. The accessory nerve.

The two most frequently seen neck swellings are

1. Lymph node enlargement.
 - Acute lymphadenitis is easily diagnosed.
 - The commonest cause of chronic lymphadenitis is the non-specific type.
 - Keep in mind the possibility of more serious causes like TB, Hodgkin's disease and secondaries.
 - Secondaries may arise from hidden malignancies
 - Nasopharynx
 - Pyriform fossa
 - Nasal sinuses
 - Thyroid papillary carcinoma
2. Thyroid enlargement (goitre).

Variations of radical block neck dissection

Suprahyoid block dissection

Bilateral dissection is restricted to the lymph nodes above the level of the hyoid bone. The submandibular salivary glands are also removed because of their close proximity to these nodes.

Indications

- Carcinoma of the lower lip.
- Sometimes in cases of early carcinoma of the tip of the tongue and floor of the mouth.

Modified radical block dissection

The operation is similar to radical block neck dissection but with preservation of the internal jugular vein and the sternomastoid muscle.

Indications

- Thyroid cancer.
- Bilateral block neck dissection. Preservation of one jugular vein on the less affected side is necessary to provide adequate venous drainage from the brain.

PHARYNX AND LARYNX

Pharynx

Anatomy of the pharynx

The pharynx is a wide muscular tube, about 5 inches long that extends from the base of the skull to the level of the 6th cervical vertebra.

Divisions (Fig. 25.1)

- **The nasopharynx** above the soft palate.
- **The oropharynx** from the inferior surface of the soft palate to the lingual surface of the epiglottis.
- **Laryngopharynx** from the epiglottis to the inferior border of the cricoid cartilage which is opposite the body of the 6th cervical vertebra. The laryngopharynx is further subdivided into the epilaryngeal and postcricoid areas.

Layers

The wall of the pharynx is lined from within outwards by mucosa, submucosa, pharyngo-basilar fascia, pharyngeal muscles and bucco-pharyngeal fascia.

Musculature

The pharyngeal musculature is composed of 3 pairs of muscles which lie in the sides and the posterior wall. These are the superior, middle and inferior constrictor muscles. The inferior constrictor muscle is composed of a lower part of transversely lying fibres (cricopharyngeus) and an upper part composed of fibres which ascend with varying degrees of obliquity (thyropharyngeus). There is a weak area in the midline posteriorly between the two parts called "Killian's dehiscence". The constrictor muscles are supplied by the pharyngeal plexus of nerves, the inferior constrictor receiving additional twigs from the external and recurrent laryngeal nerves.

Upper oesophageal sphincter

Under normal conditions the cricopharyngeus is closed to prevent air entry into the oesophagus. During swallowing the thyropharyngeus muscle contracts while the cricopharyngeus relaxes to allow the passage of food.

Retropharyngeal abscess

Acute retropharyngeal abscess

Clinical features. This is due to suppurative inflammation in the retropharyngeal lymph nodes which usually occurs as a complication of tonsillitis. It is commonest in children

CHAPTER CONTENTS

- Pharynx
 - Anatomy
 - Retropharyngeal abscess
 - Pharyngeal diverticulum
 - Tumours of the pharynx
- Larynx
 - Tracheostomy

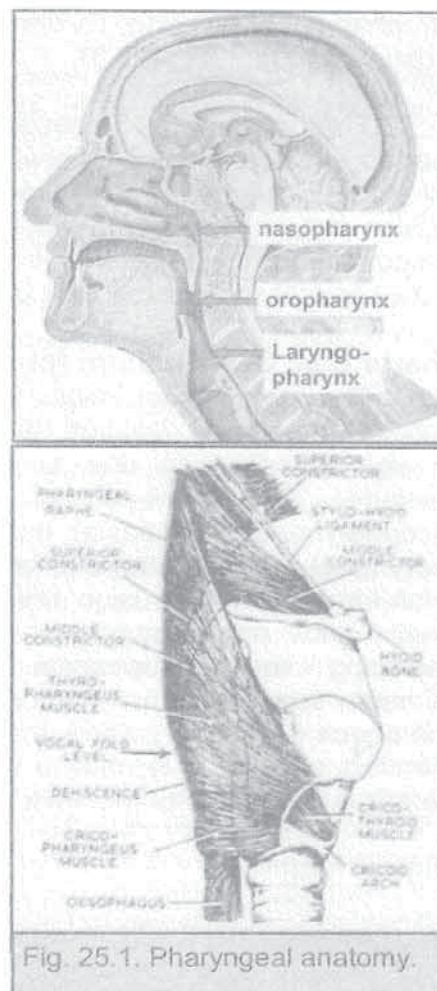


Fig. 25.1. Pharyngeal anatomy.

under the age of 4. The neck is held rigidly, saliva dribbles from the mouth, and feeds are regurgitated.

The posterior wall of the pharynx is bulging, and a fluctuant swelling may be felt digitally.

Treatment. Under general anaesthesia and using a cuffed endotracheal tube, a pair of forceps guided by the finger is thrust into the abscess cavity (Fig. 25.2). Pus is evacuated and antibiotic therapy is prescribed.

Chronic retropharyngeal abscess

Clinical features. This is a cold abscess usually due to tuberculosis of the cervical spine, and rarely due to tuberculous adenitis of the retropharyngeal lymph nodes. There is often a fluctuant swelling at the posterior border of the sternomastoid muscle, in addition to the retropharyngeal swelling. Evidence of cervical caries is usually present (Fig. 25.3).

Treatment. The abscess should never be opened into the mouth, otherwise secondary infection will occur. The pus may be evacuated through a small incision behind the sterno-mastoid and the wound is closed without drainage. The treatment of tuberculosis of the cervical spine follows the usual lines.

Pharyngeal diverticulum (pharyngeal pouch)

The diverticulum originates as a protrusion of mucous membrane through the Killian's dehiscence (pulsion diverticulum) due to raised intrapharyngeal pressure, from failure of relaxation of the cricopharyngeus (achalasia) during contraction of the pharyngeal muscles above it, in the act of swallowing. Once formed, the protrusion fills with food at every meal, and progressively, increases in size, displacing the oesophageal opening, which ultimately lies high in the anterior wall of the pouch. The pouch descends towards the mediastinum, but often turns outwards, usually to the left to appear in the posterior triangle of the neck (Fig. 25.4)

Clinical features

- The patient is usually an elderly, and the condition is twice as common in men as in women.
- In early stages the diverticulum is usually asymptomatic.
- Occasionally, it gives rise to symptoms identical to those of a foreign body in the throat.
- In long-standing cases there is history of dysphagia, recently aggravated and associated with regurgitation of undigested food after meals, eructation of gas and gurgling noise in the neck.
- Sometimes, there is an irritable cough from overflow of food into the larynx.

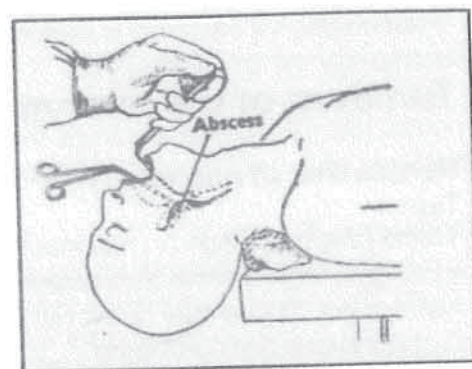


Fig. 25.2. Drainage of acute retropharyngeal abscess.

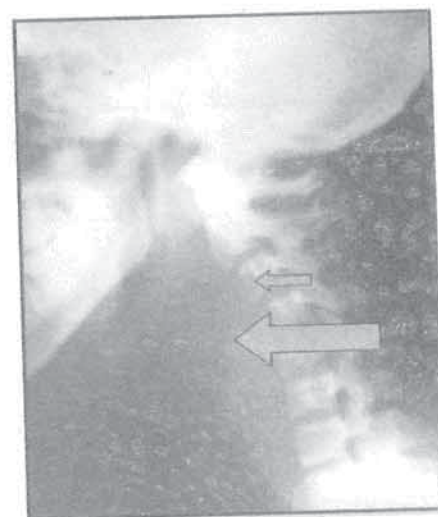


Fig. 25.3. Chronic retropharyngeal abscess. X-ray shows

1. Wide space between tracheal air and cervical spine (big arrow).
2. Osteolytic lesion of 3rd cervical vertebra (small arrow).
3. Cervical spine kyphosis due to irritation and spasm of prevertebral muscles.

- In about one-third of cases, the pouch is large enough to form a visible swelling in the neck, which enlarges when the patient drinks and can be emptied by pressure.
- In advanced cases, progressive loss of weight occurs and may lead to cachexia.

Diagnosis

Barium swallow may show a bulge in the posterior wall of the pharynx in early cases, but in late cases there is a flask shaped pouch (Fig. 25.4), sometimes with a fluid level.

Oesophagoscopy is not recommended as it may perforate the fundus of the pouch.

Manometric studies may be helpful.

Treatment

When the pouch is of a large size, surgery is recommended. The diverticulum is excised, the neck is closed in two layers and a cricopharyngeal myotomy is performed.

Tumours of the pharynx

Neoplasms of nasopharynx

Types

These may be benign or malignant.

1. Benign. Angiofibroma may cause massive bleeding.
2. Malignant. These may be either

○ Squamous cell carcinoma	70%
○ Lymphoma	15%
○ Lymphoepithelioma	10%
○ Mixed salivary tumours	5%

There is a possible role for Epstein-Barr virus in the aetiology of nasopharyngeal carcinoma. About half of nasopharyngeal malignant neoplasms arise in the lateral wall in the supratonsillar fossa.

Clinical features

1. Nasal symptoms: Early there are slight intermittent epistaxis and nasal tone speech. Later, the patient has nasal obstruction and a post-nasal discharge. Deformity of the nose does not usually occur.
2. Aural symptoms: Unilateral deafness or secretory otitis media occurs due to obstruction of the Eustachian tube.
3. Metastatic cervical lymph nodes: These may be the first presentation in 40% of patients, leading to enlargement of the upper deep cervical lymph nodes which may be bilateral. These nodes may be diagnosed clinically as malignant lymphomas.
4. Paralysis of cranial nerves Due to proximity of the neoplasm to the skull base, one or more of the cranial nerves may be involved, Involvement of the trigeminal nerve may cause pain in the side of the head.

Investigations

- Examination of the nasopharynx and biopsy.
- CT scan can reveal the extent of the tumour and invasion of the base of the skull.

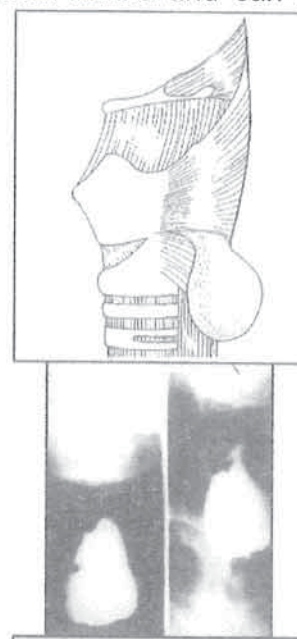


Fig. 25.4. Pharyngeal diverticulum. Barium swallow shows the pouch.

Treatment

Supervoltage external irradiation to the primary tumour and to the lymph nodes as far down as the clavicle. Surgery is not recommended as the tumour is very inaccessible and there is usually bilateral enlargement of the upper deep cervical lymph nodes up to the base of the skull.

Neoplasms of oropharynx

Types

- **Benign:** Venous malformation is treated by sclerosing solutions, laser or cryosurgery.
- **Malignant:** Squamous cell carcinoma.

Clinical features

1. Early there is slight discomfort or pain, the pain may be referred to the ear via the tympanic branch of the glossopharyngeal nerve (Jacobson's nerve).
2. Dysphagia.
3. Blood tinged sputum.
4. Metastases to the cervical lymph nodes.

Treatment

Radiotherapy or pharyngectomy.

Neoplasms of laryngopharynx

Sites and clinical picture

1. Epilaryngeal tumours

- These, include tumours of the epiglottis, aryepiglottic fold, pyriform fossa (Fig. 25.5) or the lateral wall of the pharynx.
- More in males at the age of 50-60 years.
- The tumour is either of the ulcerative or the papillary type.
- The tumour remains symptomless for a long time and the patient may present with cervical lymph node metastases.
- There may be sore throat, foetid discharge, dysphagia, or change in the quality of phonation.
- Investigations include laryngoscopy and biopsy.

2. Post-cricoid carcinoma

- More common in females about the age of 40, the tumour may occur on top of Plummer Vinson's syndrome.
- Usually it involves the anterior wall of the hypopharynx at the level of cricoid cartilage.
- The early symptom is pain in the throat referred to the side of the neck or the ear due to stimulation of the auricular branch of the vagus (Arnold nerve).
- Foul smell of breath and loss of weight.
- Dysphagia is late.
- Examination reveals forward bulging of the thyroid cartilage and trachea. The alae of the thyroid cartilage may spread outwards. There may be loss of laryngeal click.



Fig. 25.5. Carcinoma of pyriform fossa.

Investigations

- Barium swallow.
- Direct pharyngoscopy and biopsy.

Treatment

- Operable and fit patients should have total laryngopharyngectomy with block dissection of lymph nodes. The stomach is mobilized and pulled up to the neck to be anastomosed to the pharyngeal stump. The patient will be left with a tracheostomy. Another method to replace the pharynx is pectoralis major myocutaneous flap.
- Inoperable and unfit patients are treated by external irradiation.

Larynx

Tracheostomy

Indications

1. To relieve obstruction of the upper air passages due to
 - a. Foreign bodies.
 - b. Cedema of the glottis which may occur secondary to acute laryngitis, laryngeal diphtheria, corrosive swallowing and angioneurotic oedema.
 - c. Bilateral abductor paralysis of the vocal cords secondary to recurrent laryngeal nerves injury.
 - d. Carcinoma of the larynx.
 - e. Injuries of the larynx.
 - f. Chronic stenosis following tuberculosis.
2. To allow assisted or positive pressure ventilation especially if needed for prolonged periods, as an endotracheal tube cannot be left in place for more than 10 days. In these patients tracheostomy offers the patient two more advantages; reduction of anatomical dead space and effective suction of tracheobronchial secretions.
 - a. Unconsciousness following head injuries.
 - b. Extensive faciomaxillary injuries.
 - c. Flail chest wall following thoracic injuries.
 - d. Coma due to poisoning.
 - e. Tetanus.

Technique

In contrast to the traditional old teaching, tracheostomy is not quite an emergency operation that is to be done at the seen of an accident, nor in a hurry in the emergency room. The operation is a semi-emergency one that is to be done under optimal conditions in the operating theatre. For extreme emergencies there are faster and simpler alternatives that will be mentioned later.

The steps of the operation are

- General anaesthesia: If a skilled anaesthetist is unavailable, local anaesthesia can be used.
- The head and neck are extended.
- The incision is either a transverse one in the lower neck, or a strictly midline one reaching from the cricoid cartilage to the suprasternal notch.

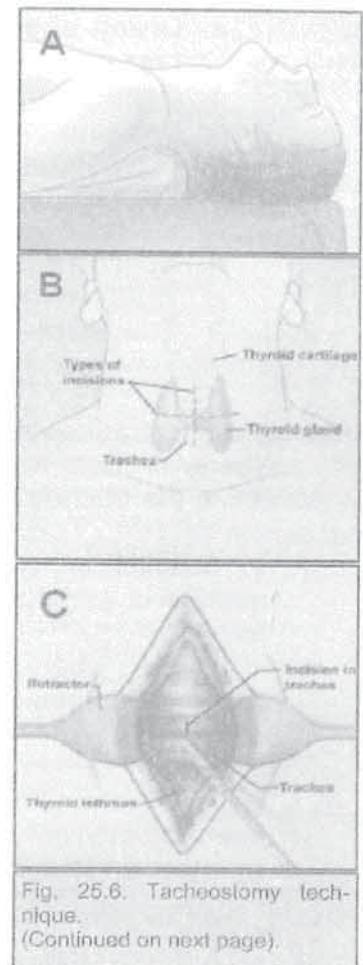


Fig. 25.6. Tracheostomy technique.
(Continued on next page).

- The pretracheal muscles are separated in the middle line to expose the thyroid isthmus which is clamped, and divided.
- A narrow ellipse is removed from the anterior wall of the second, third, and fourth tracheal rings and a suitable sized tracheostomy tube is inserted. For the purpose of mechanical ventilation a cuffed tube is used in order to prevent air leak.
- The wound is closed and the tube is fixed in place by straps.

Postoperative care

- Whenever indicated, the tube is connected to a ventilator.
- Frequent suction of secretions.
- Humidification of the inspired air to avoid dryness of the air passages and the secretions which become difficult to expel and aspirate. This is achieved either by nebulizers, or by simply applying a single layer of wet gauze to the opening of the tube.

Complications

1. Early

- a. Drying of secretions and obstruction of the tube.
- b. Damage of the tracheal wall from prolonged pressure by the cuff. This may lead to haemorrhage.
- c. Displacement of the tube into the tissues of the neck.
- d. Surgical emphysema.
- e. Wound infection

2. **Late.** Subglottic stenosis occurs if the tracheal incision involves the first tracheal ring.

Alternatives to tracheostomy

Whenever there is an extremely urgent need to get an access to the trachea the following techniques are fast and simple

1. In hospital or in an ambulance, the preferred method is endotracheal intubation either orally or nasally.
2. In places where such facility is not available, cricothyroidotomy offers a good temporary airway. The cricothyroid membrane is easily felt between the thyroid and the cricoid cartilages. A knife is used to cut through the skin and the membrane. Any available tube is then pushed inside the trachea. A less effective alternative is to insert 2-3 wide bore needles in the cricothyroid membrane. When the patient reaches a hospital the cricothyroidotomy is replaced by either endotracheal intubation, or a tracheostomy.

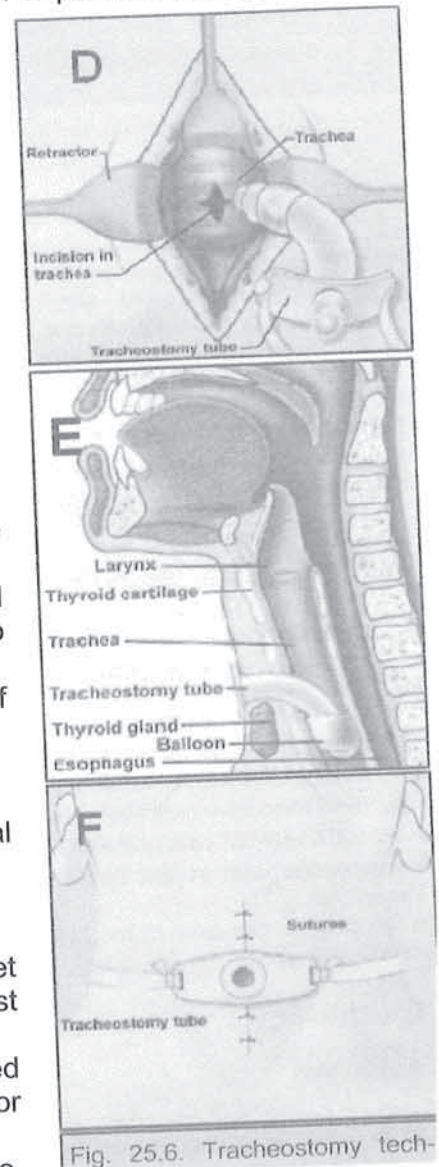


Fig. 25.6. Tracheostomy tech-

ENDOCRINE SURGERY

The endocrine glands are composed of the pituitary gland (the maestro), thyroid gland, parathyroid glands, suprarenal glands and the endocrine parts of pancreas and gonads. The functions of these glands are regulated by feeding pituitary hormones except for autonomous hyperfunction mostly of tumours and rarely hyperplastic glands.

The diagnosis of endocrine hyperfunction became easy through immunoassay. Localization of the hyperfunctioning site became also possible through modern imaging and isotopic studies.

APUD cells

APUD is an acronym that stands for the ability of these cells for Amine Precursor Uptake, and then their Decarboxylation to produce a variety of hormones. These are endocrine cells of ectodermal origin arising from the neural crest and migrate during intrauterine life to different organs in the body. Examples of these cells include

- Pancreatic islet cells that secrete glucagon, insulin, and gastrin.
- Anterior pituitary cells that secrete ACTH.
- Adrenal medullary cells that secrete catecholamines.
- Parafoollicular "C" cells of the thyroid that secrete calcitonin.
- Argentaffin cells in the bowel (Kulchitzki cells) that secrete serotonin.

In this chapter disorders of the APUD cells of the thyroid, parathyroid and adrenal glands are discussed.

Apudomas and multiple endocrine neoplasia syndrome

Tumours, benign and malignant, as well as hyperplasia can affect the APUD cells producing functioning endocrine tumours known as apudomas. The neoplasia may be confined to one organ or may involve different APUD cells in different organs producing the multiple endocrine neoplasia (MEN) syndrome. It is also known as multiple endocrine adenoma (MEA) syndrome. There are two main distinct types of the MEN syndrome. The parathyroid cells, though not members of the APUD system, are affected by hyperplasia in both types of the syndrome.

CHAPTER CONTENTS

- APUD cells
- Thyroid gland
- Thyroid-Developmental anomalies
- Goitre classification
- Thyroiditis
- Simple goitre
- Thyrotoxicosis
- Hypothyroidism
- Thyroid neoplasms-Classification
- Thyroid Cancer
- Solitary thyroid nodule
- Parathyroid glands-Basic knowledge
- Hyperparathyroidism
- Hypoparathyroidism
- Adrenal glands-Basic knowledge
- Adrenal neoplasms
- Cushing's syndrome
- Hyperaldosteronism
- Pheochromocytoma

MEN

Inherited condition.

Autosomal dominant.

All family members should be screened.

MEN I (Wermer's syndrome)

1. Parathyroid hyperplasia.
2. Islet cell tumours of the Pancreas (gastrinoma, or insulinoma)
3. Anterior Pituitary tumours (usually chromophobe adenoma)

All start with P.

MEN II

- MEN Ia (Sipple's syndrome)

1. Medullary carcinoma of the thyroid.
2. Pheochromocytoma.
3. Parathyroid hyperplasia.

- MEN IIb

1. Medullary carcinoma of thyroid.
2. Pheochromocytoma
3. Mucosal neuromas involving the lips, tongue and inner aspect of eyelids.

Thyroid gland

Embryology (Fig. 26.1)

- The thyroid appears embryologically by the third week of life, as a proliferation of epithelial cells in the floor of the developing pharynx (the thyroglossal duct) at a point indicated by the foramen caecum, a depression at the base of the tongue.
- As the thyroid primordium descends, it unites with the ultimobranchial body which arises on either side from a diverticulum of the fourth pharyngeal pouch. Parafollicular (C-cells) are derived from the neural crest and reach the thyroid via the ultimobranchial body. After the thyroid reaches its definitive location in the lower neck, the connecting thyroglossal duct undergoes obliteration.

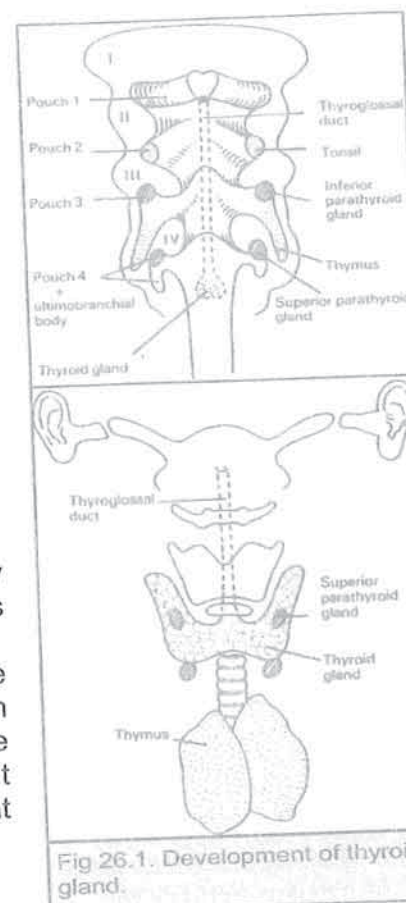


Fig 26.1. Development of thyroid gland.

Anatomy

General features and position (Fig. 26.2)

The normal thyroid gland is a brownish-red highly vascular endocrine gland consisting of two lobes connected across the midline by an isthmus.

Each lobe extends superiorly up to the oblique line of the thyroid cartilage and inferiorly to the fourth or fifth tracheal rings, while the intervening isthmus overlies the second and third tracheal rings. The gland weighs about 20-25 gm in the adult and enlarges physiologically at puberty and during menstruation and pregnancy.

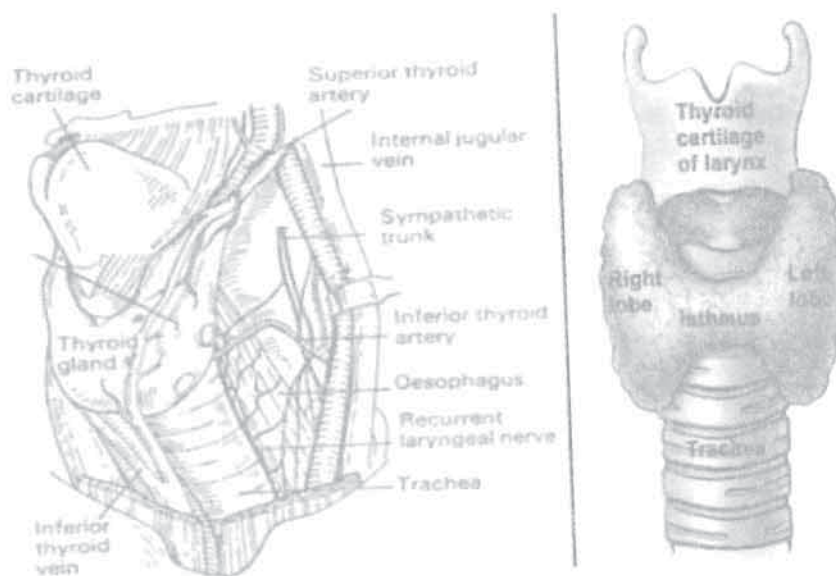


Fig. 26.2. Anatomy of the thyroid gland

Relations (Fig. 26.3)

- **Medially** each lobe is moulded over the larynx and trachea.
- **Superficially**, the gland is covered by the sternothyroid, the sternohyoid and inferiorly by the anterior border of the sternomastoid.
- **Superiorly** the gland overlies the cricothyroid muscle. The external branch of the superior laryngeal nerve passes deep to the upper pole of the gland on its way to supply this muscle.
- **Posteromedially** the thyroid lobe is related to the tracheoesophageal groove in which runs the recurrent laryngeal nerve.
- **Posterolaterally**, the thyroid is in contact with the carotid sheath. The parathyroid glands are usually related to the posterolateral surface of the thyroid gland.
- **Pretracheal fascia.** The thyroid gland is enclosed in a sheath of the pretracheal fascia, which is attached above to the arch of the cricoid and oblique line of the thyroid cartilage, that is why the gland moves with the larynx in all its movements.

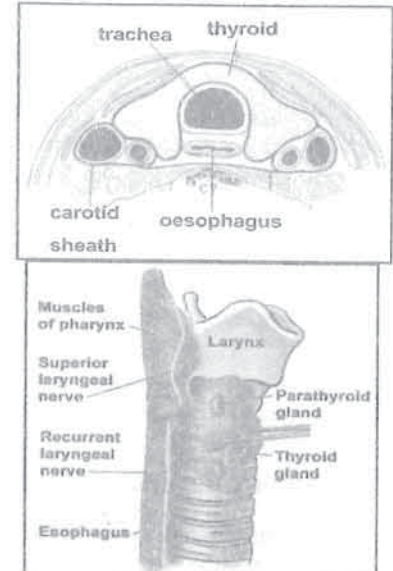


Fig. 26.3. Relations of thyroid

Arteries

The arterial blood supply of the thyroid gland is very rich.

1. Superior thyroid arteries, branches of the external carotids.
2. Inferior thyroid arteries arising from the thyrocervical trunks of the subclavian arteries. The recurrent laryngeal nerve comes into direct relationship with the terminal branches of the inferior thyroid artery being posterior, but may pass anterior to or between the arterial branches (Fig. 26.4).
3. An additional minimal blood supply comes via the "thyroidaeima" artery, being variable in size, originating from the innominate artery or the aortic arch, it ascends upwards anterior to the trachea reaching the lower border of the gland.
4. The esophageal arteries also supply the gland.

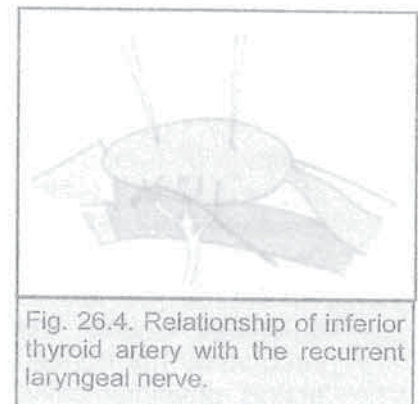


Fig. 26.4. Relationship of inferior thyroid artery with the recurrent laryngeal nerve.

Veins

Thyroid veins begin to form on the surface of the gland

1. The superior thyroid vein drains into the internal jugular vein coursing with the corresponding artery.
2. The middle thyroid vein passes in most instances as a well defined trunk, draining into the internal jugular vein, and can be quite short if the gland is enlarged.
3. The inferior thyroid veins pass from the lower pole of each lobe and course to the left brachio-cephalic trunk (innominate vein).

Lymphatic drainage

An extensive network of lymphatics lies within the gland.

- The subcapsular plexus drains principally to the juxta-thyroid lymph nodes, i.e., prelaryngeal, pretracheal lymph nodes and to lymph nodes along the recurrent laryngeal nerve, thence to the deep cervical and mediastinal lymph nodes.
- Some lymphatics pass directly to the deep cervical lymph nodes.

Physiology

The functioning unit of the thyroid gland is the lobule, supplied by a single arteriole and consisting of 20-40 follicles, which are lined by cubical epithelium. The resting follicle contains colloid in which thyroglobulin is stored (Fig. 26.5).

Thyroid hormone synthesis

The normal thyroid gland is able to concentrate iodide from the blood at a rate of about 2 ug per hour.

Once within the thyroid follicular cells, the iodides are incorporated into the synthesis of the hormones Tri-iodo-thyronine (T_3) and thyroxine (T_4), the process being controlled by several enzymes in distinct steps.

1. Trapping of inorganic iodide from the blood. This is an active process dependent on ATP.
2. Oxidation of iodide into iodine by the peroxidase enzyme
3. Binding of iodine with tyrosine by the tyrosinase enzyme to form mono and diiodotyrosines (organification).
4. Coupling of mono-iodotyrosines and di-iodotyrosines to form T_3 and T_4 which unite with thyroglobulin and are stored in the follicles.
5. Release of T_3 and T_4 into the circulation upon need. The major fraction of T_3 and T_4 in the blood is bound to plasma proteins (albumin, prealbumin and thyroid binding globulins). A minute fraction of T_3 and T_4 is present in a free form and this represents the functionally active fraction. (Free T_4 0.03-0.04% and free T_3 0.2-0.5% of the total circulating hormones respectively), T_3 is the quick acting, physiologically important hormone, being also produced by peripheral conversion from T_4 .
6. Few mono and diiodotyrosines are not coupled to form T_3 or T_4 . They are deiodinated by dehalogenase enzyme. The released iodine is reused in thyroid hormone synthesis instead of being wasted.

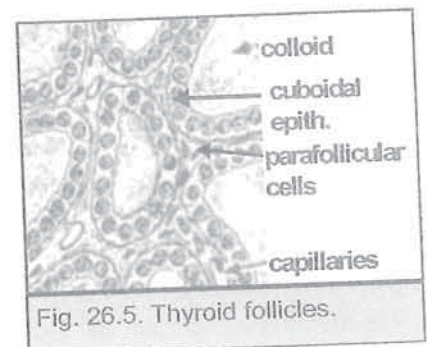


Fig. 26.5. Thyroid follicles.

T_3 is the most active form of thyroid hormone.

Hypothalamo-pituitary-thyroid axis (Fig. 26.6)

Synthesis and liberation of thyroid hormones are controlled by the thyroid stimulating hormone (TSH) produced by the anterior pituitary. Its level assumes an inversely proportional relation to the level of circulating thyroid hormones (feed back mechanism). Thyrotropin releasing hormone (TRH) produced by the hypothalamus is a further stimulant to its production.

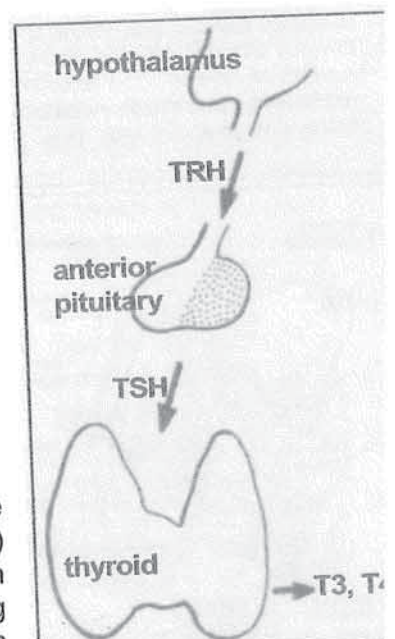


Fig. 26.6. Hypothalamo-pituitary thyroid axis.

Tests of thyroid function

1. **Measurement of serum TSH.** The test is routinely performed with a normal range of 0.5-5 Mu/l. It is now possible to do **ultrasensitive TSH** and this test now is the most sensitive for the assessment of the thyroid function. Elevated level is the earliest test to diagnose hypothyroidism.
2. **Measurement of thyroid hormones in the serum:**
 - a. Total serum thyroxine (T_4) [55-150 nmol/l].
 - b. Total Tri-iodothyronine (T_3) [1.2-3.1 nmol/L].
 - c. Free serum thyroxine (T_4) [8-26 pmol/l].
 - d. Free serum Tri-iodothyronine (T_3) [3-9 pmol/l].

Total serum T_4 and T_3 are no reflection of the free active hormones. They are affected by fluctuations in the levels of the thyroxine binding proteins (namely thyroid binding globulin and pre-albumin). False elevated levels of total serum T_3 or T_4 accompany pregnancy and oral contraceptive intake. On the other hand false low results occur with hypoproteinaemia, e.g. nephrotic syndrome.
3. **Thyrotropin releasing hormone (TRH).** Intravenous 500 ug TRH is administered and the level of TSH is measured and compared to the baseline level. This test is sometimes used to assess patients with borderline hyperthyroidism.
4. **Thyroid antibodies** These are raised in Hashimoto's thyroiditis and Graves' disease.
5. **Tests using radioactive ^{123}I**
 - a. Radioactive iodine uptake test: Following an oral dose of 5 uCi, the percentage of uptake of radioactive material by the thyroid gland is measured at 4 hours (The normal uptake is 11-55%). The level of radioactive thyroxine bound to plasma proteins in the blood is measured at 24 and 48 hours. In hyperthyroidism a high counting rate is detectable over the thyroid gland after 4 hours, followed by an elevated plasma protein ^{123}I at 24 or 48 hours.
 - b. Thyroid scanning. After a tracer dose of ^{123}I or Technetium⁹⁹ pertechnetate (^{99m}Tc) a scan of the thyroid gland is obtained. It allows evaluation of the functional activity of the different areas of the gland, whether normal (warm) or hyperactive (hot) or non functioning (cold). The principal value is in identifying an autonomous toxic nodule whether solitary or a part of toxic multinodular goitre. Risk of malignancy in cold nodules is 15-20%.

Thyroid - Developmental anomalies (Fig. 26.7)

Thyroglossal cyst

Pathology. This is a midline tubulodermoid cyst arising in thyroglossal duct remnant. The thyroglossal duct can assume a variable relationship to the hyoid bone; behind or in front or passing through the body of the bone. The cyst may occur at any level from the foramen caecum of tongue to the suprasternal notch (Fig. 26.8), usually in the midline just below the hyoid bone except at the level of the thyroid cartilage, where it is displaced usually to the left.

Clinically

- The cyst may present at any age, but is most common during childhood.
- It appears as a painless cystic swelling that moves up and down on swallowing and on protrusion of the tongue (due in its relation to the hyoid bone).

- There may be a palpable track extending from the hyoid bone upwards towards the tongue.
- The wall of the cyst is rich in lymphatics which may communicate with the cervical lymph nodes, so infection may be the presenting symptom and may even lead to fistula formation when an infected thyroglossal cyst ruptures or is incised.

Treatment is by surgical excision of the cyst and its associated tract entailing removal of the middle third of the body of the hyoid bone (Sistrunk's operation). If the excision is incomplete, recurrence in the form of thyroglossal cyst or fistula may result.

Thyroglossal fistula

Aetiology. This is an acquired fistula that is caused either by

- Infection of a thyroglossal cyst leading to rupture
- Inadequate removal of the cyst.

Pathology. The track is lined by columnar epithelium, discharges mucus, and presents with recurrent inflammation.

Clinical features. The opening of the fistula is near the midline on the left side with a crescentic fold. A track can usually be palpated extending from this opening upwards to the hyoid bone (Fig. 26.8).

Treatment is by excision of the fistula together with the central part of the hyoid bone. If there is an upward extension beyond the hyoid bone it should be followed up even to the foramen caecum of the tongue.

Lingual thyroid

Pathology. Failure of descent of the thyroid can lead to an intralingual development of the gland.

Clinically, patients may present with a tongue swelling with obstructive manifestations i.e. dysphagia, difficult breathing or change in the quality of voice or even haemorrhage.

Investigations. Thyroid scans will establish the absence of thyroid tissue in the normal site in the neck.

Treatment options include full replacement with L-thyroxine to induce reduction in size, surgical extirpation or radioactive-iodine ablation.

Goitre classification

This term means generalized thyroid enlargement. Based on the aetiological and clinical picture goitre may be classified into

1. **Inflammatory goitre (Thyroiditis)**
 - a. Acute bacterial (rare).
 - b. Subacute thyroiditis (De Quervain's disease).
 - c. Chronic, e.g. tuberculosis or syphilis (rare).
 - d. Autoimmune goitre as Hashimoto's thyroiditis or Riedel's thyroiditis.

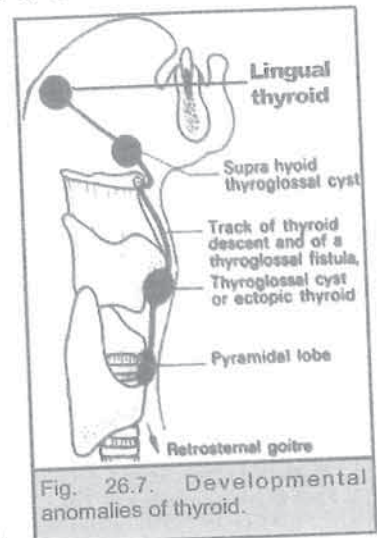


Fig. 26.7. Developmental anomalies of thyroid.

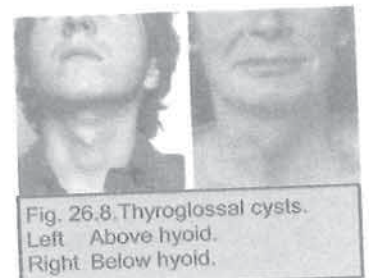


Fig. 26.8. Thyroglossal cysts.
Left Above hyoid.
Right Below hyoid.



Fig. 26.8. Thyroglossal fistula.

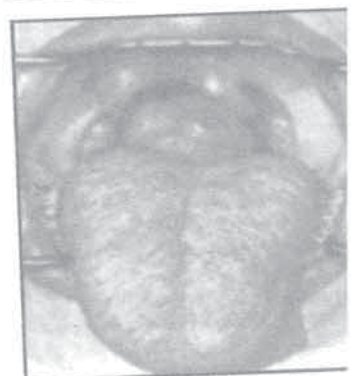


Fig. 26.9. Lingual thyroid

2. **Toxic goitre** means thyroid enlargement accompanied by the clinical picture of hyperthyroidism.
3. **Neoplastic goitre** may be benign or malignant.
4. **Simple goitre** means thyroid enlargement, not accompanied by inflammatory, toxic or neoplastic criteria. This may be (a) Simple physiological goiter, (b) Simple colloid goiter, (c) Simple nodular goitre.

Thyroiditis

Table 26.1. Types and aetiology of thyroiditis

Thyroiditis	Cause
Acute	Pyogenic bacteria, rare
Subacute	? Viral
Chronic	T.B., rare
Hashimoto's	Autoimmune
Riedle's	

Subacute thyroiditis (granulomatous thyroiditis, De Quervain's thyroiditis)

- The aetiology of the condition is controversial, being most probably a viral infection.
- The condition has a moderately abrupt onset, often following an upper respiratory infection. The thyroid becomes congested, swollen with a firm irregular pattern and slightly tender. It is accompanied by neck pain with fever and malaise.
- Elevation of ESR with a normal or depressed leucocytic count is a feature. Iodine-uptake by the gland is depressed in the presence of a slight elevation of serum T4.
- The condition is characterized by remissions and exacerbations over a period of up to few months, but usually it is self-limited. A rapid response to oral prednisone is diagnostic, and a course of the drug may be required for few weeks. In the occasional severe cases, anti-inflammatory drugs are beneficial.

Autoimmune thyroiditis (Hashimoto's thyroiditis)

The condition is the most common form of thyroiditis, which usually affects females at menopause.

Aetiology. It is supposed to be due to the presence of antibodies to thyroid antigens.

Pathologically there is infiltration of the thyroid gland by lymphocytes and plasma cells. Eventually the acini are replaced by this infiltrate leading to the development of myxoedema. It exhibits a variable onset being insidious and asymptomatic in some cases or sudden and painful in others.

Clinical features. The goitre is usually lobulated (flat-topped nodularity), the condition being diffuse or localized to one lobe. It may be large or small, soft, rubbery or firm in consistency. Mild hyperthyroidism may be present initially but hypothyroidism is inevitable.

Investigations. Serum titres of antimicrosomal and anti-thyroglobulin antibodies are elevated. Nevertheless, differential diagnosis from nodular goitre or carcinoma may require fine needle or tru-cut needle biopsy.

Treatment entails full replacement dosage of thyroxine. Surgical treatment may be needed for large-sized goitres or suspected development of lymphoma which is occasionally associated with Hashimoto's goitre or develops later in the disease.

Riedel's thyroiditis

- This is a rare condition, accounting for 0.5% of goitres.
- It presents as a hard woody transformation of thyroid tissue due to extensive fibrosis which extends even beyond the gland. It may occur in association with retro-peritoneal fibrosis. Some authors consider this disease as an autoimmune disorder, others consider it as a collagen disease.
- Hypothyroidism is usually present. The differentiation from anaplastic carcinoma may mandate an open biopsy where a wedge of the isthmus is removed to free the trachea.

Simple goitre – endemic or sporadic

This term is applied to thyroid enlargement not belonging to the inflammatory, thyrotoxic or neoplastic groups.

Aetiology

Simple goitre results from stimulation of the thyroid gland by elevated levels of TSH, as a sequence of an absolute or relative decrease in the circulating thyroid hormones (Fig. 26.10).

1. **Iodine deficiency.** This may be absolute or relative. Normal daily requirements of iodine is 100-125 ug. Simple goitre is endemic in regions with low iodide content of the water and food as in mountainous regions or low-land areas with a soil lacking iodide. In Egypt oasis areas are famous for endemic goitre. Relative deficiency of iodine occurs during periods of stress. Females during the menarche, pregnancy and lactation are susceptible to this problem.
2. **Enzymatic deficiency.** This is responsible for many cases of sporadic goitre, often being associated with a positive family history, reflecting an underlying genetic defect.. **Pendred's** syndrome is an example being due to deficiency of peroxidase enzyme which transforms inorganic iodides to organic iodine. In this syndrome there is goitre, deafness and mutism. Patients develop goitre at a young age, sometimes several brothers or sisters are affected. The patient is liable to get recurrent goitre after surgery. Deficiency of dehalogenase enzyme is also responsible for sporadic goitre.
3. **Goitrogens.** Intake of goitrogens results ultimately in goitre formation. These include thiocyanates in cabbages, drugs as para-amino salicylic acid and antithyroid drugs.

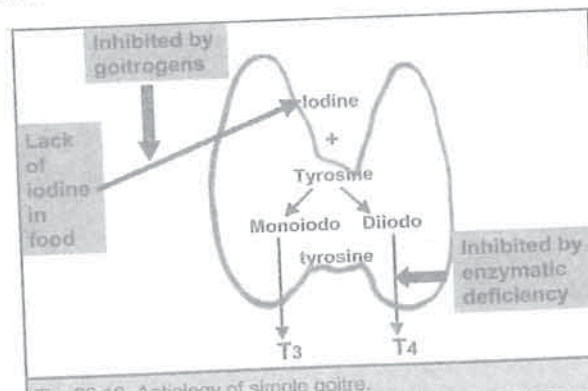


Fig. 26.10. Aetiology of simple goitre.

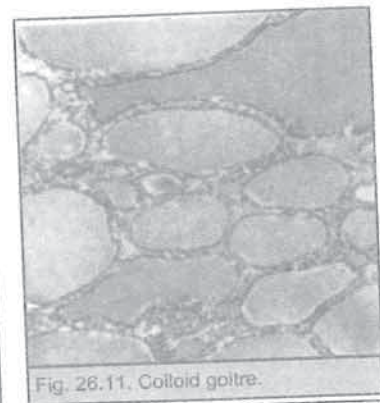


Fig. 26.11. Colloid goitre.

Stages of goitre formation

1. Deficient synthesis of thyroid hormone leads to persistent TSH elevation that produces diffuse homogenous hyperplasia; all follicles are active with uniform iodine uptake. This stage represents diffuse hyperplastic (physiological) goitre and is reversible on cessation of its stimulus i.e. increased TSH. If the iodine requirements are satisfied, the follicles undergo involution or even hyperinvolution where the follicles are distended with colloid and are lined by flattened epithelium.
2. Repeated fluctuations in the level of TSH due to repeated cycles of stress and strain produce heterogeneity of the gland, with areas of active follicles and others with inactive follicles.
3. When the iodine requirements are satisfied the follicles undergo hyperinvolution and become large in size, lined by flattened epithelium and full of colloid. Clinically this is the simple colloid goiter (Fig. 26.11).
4. As a result of recurrent hyperplasia and hypervascularity, haemorrhages occur producing central necrosis in the lobules. These ultimately coalesce into nodules containing iodine-free colloid or masses of new inactive follicles. The nodules are enclosed in delicate connective tissue, most nodules are inactive, the active follicles being present in the internodular tissue. This stage is called nodular goiter.
5. Other factors different from TSH have also been implicated in the aetiology of nodular goitre, e.g. immunoglobulins. These by acting on oversensitive cells and/or receptors produce a nodular goitre with marked heterogeneity of structure and function between adjacent follicles.

In a single lobule all follicles behave similarly. Active follicles are small, lined by tall columnar cells and they are empty. Inactive follicles are large, lined by flattened epithelium and are full of colloid.

Types of simple goitre

Simple thyroid enlargement is more common in females as they are more liable to repeated stresses due to the menstrual cycles, pregnancy and lactation.

1. Simple physiological (diffuse hyperplastic) goitre.

The patient presents with a diffuse, soft, homogenous enlargement of the whole gland, which may appear in childhood in endemic regions. In sporadic cases (non-endemic) its appearance corresponds to an increased metabolic demand, e.g. puberty or pregnancy. The condition is reversible upon disappearance of the triggering stimulus.

2. **Simple colloid goitre.** This due to hyperinvolution of diffuse hyperplastic goitre, the patient receiving large doses of iodine producing a

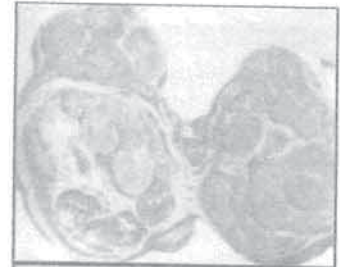


Fig. 26.12. Gross appearance of simple multinodular goitre.



Fig. 26.13. Multinodular goitre.

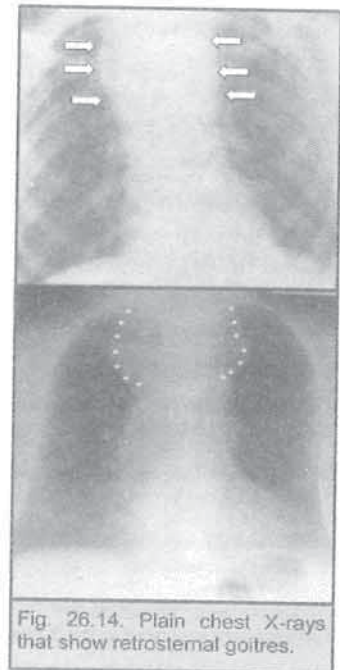


Fig. 26.14. Plain chest X-rays that show retrosternal goitres.

gland the follicles of which are lined by flattened epithelium and distended with colloid. Clinically the gland is enlarged, the surface is smooth and its consistency is soft. The cut surface of the gland has a glistening surface as the follicles are full of colloid (Fig. 26.11).

3. **Simple nodular goitre.** Usually multiple nodules are grossly present. Sometimes one of the nodules is prominent, while the rest of the nodules are not palpable and the patient is diagnosed as having a solitary thyroid nodule.

Complications of simple nodular goitre

1. Tracheal obstruction by compression. The trachea may be compressed on either side and becomes transformed into anteroposterior slit (scabbard trachea), it may be compressed anteroposteriorly, or it may be displaced to one side by marked unilateral enlargement. In long-standing nodular goitre, the tracheal rings undergo chondromalacia and the tracheal rings may collapse post-operatively.
2. Secondary thyrotoxicosis may occur in up to 30% of cases.
3. Malignancy. Occasionally follicular carcinoma may develop. The incidence is about 3%.
4. Cyst formation.
5. Haemorrhage into a nodule.
6. Calcification may occur in longstanding cases.
7. Retrosternal extension.

Clinical picture of simple nodular goitre

The condition appears early in endemic regions but may be delayed to the third decade or later in sporadic cases.

The patient's main complaint is usually the cosmetic deformity (Fig. 26.13) or some respiratory obstruction.

The thyroid gland is firm in consistency with a nodular surface. The nodules are smooth, firm and painless.

The patient is euthyroid.

Sudden enlargement with the appearance of pain and tenderness usually results from haemorrhage into a nodule.

Calcification may result in hardness and irregularity simulating malignancy.

Investigations of simple nodular goitre

- Differentiation from mildly toxic nodular goitre may require assessment of thyroid functions.
- Assessment of the titres of thyroid antibodies to exclude Hashimoto's disease may be sometimes needed.

Prevention and treatment of simple goitre

Prevention

The use of iodized table salt (potassium iodide 1 part 10,000) has proved successful for prophylaxis in endemic areas.

Treatment of diffuse hyperplastic goitre

Diffuse hyperplastic goitre is usually reversible by the use of L-thyroxine, usually 0.1 mg/day for several months to be tapered to 0.1 mg/day for several years. The patient is reassured and is advised for regular follow-up.

Treatment of multinodular goitre

Most authorities agree upon the irreversibility of multinodular goitre, thus surgical excision (thyroidectomy) is indicated for

1. Evidence of compression manifestations mainly on the trachea.
2. Cosmetic reasons.

Surgery for multinodular goitre should not be advised below the age of 25 unless very much indicated as it may be followed by recurrence. Surgery entails removing the nodular parts leaving an equivalent of 8 gm of relatively normal thyroid tissue (size of a normal lobe) on each side if feasible. Sometimes the distribution of the pathology requires removal of a whole lobe and subtotal excision of the other lobe. Post-operatively suppressive doses of L-thyroxine 0.1-0.2 mg/day are advisable to avoid recurrence in the remainder of thyroid tissue if constant iodine dietary supplementation is not continuously ascertained.

Retrosternal goitre**Pathology**

Retrosternal goitres usually arise in a normally placed thyroid gland. The sternohyoid and sternothyroid muscles prevent forward expansion, and assisted by gravity and the negative intrathoracic pressure, they direct the swelling into the mediastinum. In most cases, the intrathoracic swelling occupies the superior mediastinum.

Retrosternal goitre is classified into 3 types

1. Plunging goitres which rise with deglutition and then descend again through the thoracic inlet.
2. Mediastinal goitres which lie wholly in the chest, but are connected with the thyroid by a relatively narrow band of tissue and derive their blood supply from the thyroid vessels.
3. Intrathoracic goitres which lie wholly in the chest, completely separate from the main gland. They probably arise from congenitally misplaced thyroid tissue, deriving an independent blood supply from mediastinal vessels.

Clinical features**Symptoms**

- An intrathoracic goitre is more common in men, particularly short-necked individuals, and because it often remains symptomless for years, the patient usually presents after middle age.
- The goitre displaces and compresses the trachea causing dyspnea, which is worse at night, and is often spasmodic. It varies with the patient's position, being aggravated by any posture that reduces the thoracic inlet, such as lying down or flexion of the neck, so that many patients prefer to spend the night in a chair. Some patients are diagnosed as asthmatics.
- Sometimes there is dysphagia.

Signs

- Inspection. The innominate veins may be obstructed so that the superficial veins of the upper part of the chest are obviously dilated and there may be even cyanosis and oedema of the face and neck. The lower border of goitre cannot be seen as it lies behind the sternum.
- Palpation of the neck may reveal an enlarged thyroid gland.
- Percussion of the sternum may reveal retrosternal dullness.

Investigations

- Plain X-ray of the chest shows a shadow in the superior mediastinum (Fig. 26.14) which moves vertically with deglutition unless the goitre is firmly wedged between the structures which surround it or is intrathoracic in type. Calcification often occurs in the walls of the goitre, and may be demonstrated radiologically.
- ^{99m}Tc scan can reveal the retrosternal extension.
- CT scan of the thorax will reveal the exact level of the retrosternal extension and its anatomic relations.

Treatment

Thyroidectomy is the only line of treatment.

Resection is almost always possible via the cervical approach, rarely a median sternotomy is required. Devascularization is done via the neck from which the retrosternal portion derives its blood-supply. Special care should be exerted to avoid injury of the recurrent laryngeal nerves during delivery of the retrosternal goitre.

Toxic goitre (thyrotoxicosis = hyperthyroidism)

Common types

1. Diffuse toxic goitre (primary toxic goitre or Graves' disease), 76%.
2. Toxic nodular goitre (secondary toxic goitre or Plummer's disease), 14%.
3. Toxic nodule, 5%.

Rare causes of hyperthyroidism

1. Thyrotoxicosis factitia; due to excess intake of L-thyroxine (>0.3 mg per day).
2. Jod-Basedow thyrotoxicosis. This may occasionally occur in cases of hyperplastic endemic goitre upon the administration of large doses of iodine, the condition is temporary and very rarely permanent.
3. Subacute thyroiditis (De Quervain's disease): Mild manifestations of thyrotoxicosis may occur due to liberation of hormones from destroyed tissue.
4. Hashitoxicosis: About 5% of patients with Hashimoto's disease may have hyperthyroidism in the early stages of the disease.
5. A large mass of secondary carcinoma may be functioning.
6. Neonatal thyrotoxicosis may occur in newborns of thyrotoxic mothers or of mothers with history of thyrotoxicosis, due to transmission of thyroid antibodies across the placenta. The condition subsides in 3-4 weeks upon decline of the titre of the antibodies in the baby's serum.
7. TSH secreting adenoma of the pituitary gland.

Pathology

Diffuse toxic goitre (Graves' disease, exophthalmic goitre)

Aetiology. The disease is believed to be due to the presence of thyroid stimulating antibodies which combine with thyroid stimulating hormone receptors in the thyroid gland cells leading to the release of cAMP. This will lead to activation and increased production of T_4 .

Macroscopically, there is diffuse enlargement of the whole thyroid gland (Fig. 26.15).

Microscopic picture. There is proliferation of the epithelial lining of the acini which may be arranged in several layers. The cells are columnar, and full of



Fig. 26.15. Gross appearance of Graves' disease

granules. The acini contain less vacuolated colloid or are devoid of it (Fig. 26.16). There is marked increase in vascularity making surgery, if indicated, difficult. Extensive lymphocytic infiltration is present. Administration of iodine to these patients will diminish the vascularity and leads to storage of colloid.

Abnormal physiology. An excess of thyroid hormones produces a state of hypermetabolism that affects the whole body systems (see later). The nervous, metabolic and eye manifestations are more marked than the cardiovascular. However, some of the manifestations of Graves' disease are not due to hyperthyroidism, e.g. the exophthalmos and pretibial myxoedema.

This disease affects mainly young females, is usually of abrupt onset and is characterized by remissions and exacerbations.

Nodular toxic goitre (Plummer's disease)

- This occurs on top of a long-standing simple nodular goitre, hence affecting middle aged and elderly persons.
- It is very rarely, if ever, accompanied by extra-thyroidal manifestations, e.g. advanced exophthalmos.
- It is usually the internodular tissue that becomes hyperactive, however occasionally one or more nodules become the site of hyperactivity.
- The onset is usually insidious and may present with cardiovascular manifestations.

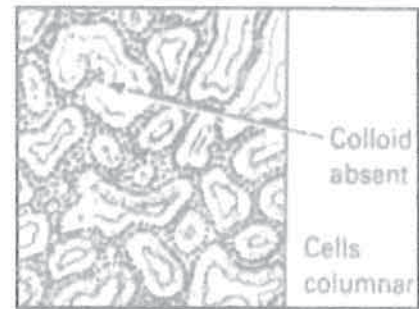


Fig. (26.16): Microscopic picture of Graves' disease

Autonomous toxic nodule

- Toxicity is due to a solitary, hyperactive, autonomous nodule (not due to thyroid antibodies).
- The toxic nodule produces suppression of TSH secretion with subsequent suppression of the remainder of the thyroid gland which becomes almost atrophic as evident on isotope scanning.

Clinical features

Most but not all symptoms and signs of thyrotoxicosis are due to excess thyroid hormones. Many systems are vulnerable to these hormones.

1. **Metabolic.** There is loss of weight in spite of increased voracious appetite. The patient complains of excessive sweating and intolerance to hot weather. The hands of the patient are warm due to vasodilation and are moist.
2. **Nervous system**
 - Symptoms: The patient complains of nervousness, bad temper, insomnia or bad dreams.
 - Signs: The patient looks tense and irritable. There may be tremors in the outstretched hands. The reflexes, e.g. the knee jerk are exaggerated.
3. **Cardiovascular system**
 - **Symptoms:** The patient complains of palpitation sometimes even at rest. There may be anginal pains. If there is heart failure, the patient complains of orthopnea, pain in the right hypochondrium and bilateral oedema of the lower limbs.
 - **Signs**

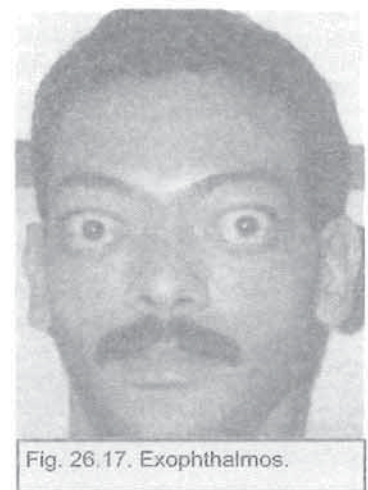


Fig. 26.17. Exophthalmos.

(a) Tachycardia with a sleeping pulse more than 90/minute.

- (b) Water hammer pulse is due to high systolic (due to increased cardiac output) and low diastolic blood pressure (due to decreased peripheral resistance).
- (c) Arrhythmias: All sorts of cardiac arrhythmias except heart block may occur. There may be multiple extrasystoles, paroxysmal atrial tachycardia, paroxysmal atrial fibrillation or persistent atrial fibrillation not responding to digoxin.
4. **Muscular.** There is usually muscular weakness with rapid exhaustion after minor effort. In severe cases proximal myopathy and muscle atrophy may occur. The proximal muscles as the shoulder and plevic girdle muscles are particularly affected.
 5. **Skin.** Patchy pigmentation or vitiligo may occur. Pretibial myoedema presents as multiple orange yellow patches of thickening of the skin over the shin of the tibia. They are due to deposition of mucin like substances under the effect of LATS.
 6. **Gastrointestinal tract** there may be diarrhoea.
 7. **Endocrine system.** Menstrual abnormalities may occur in females as menorrhagia. There may be abnormal libido, gynaecomastia or impotence in males.
 8. **Urinary system.** There may be polyuria or glycosuria.
 9. **The eyes:**

▪ Exophthalmos

- (a) True exophthalmos, implying actual protrusion of the eyeball (Fig. 26.17), is due to deposition of fluid and round cell infiltrate in the retrobulbar tissues. It is characteristic of Graves' disease. The condition is aggravated by ophthalmic vein compression, leading to lid-oedema, conjunctival injection and chemosis (Fig. 26.18). Weakness of extra-ocular muscles results in diplopia. In severe cases papilloedema, corneal ulceration and optic nerve neuropathy may occur. The severe progressive form is known as malignant exophthalmos. It is postulated that true exophthalmos is an autoimmune disease due to thyroid stimulating antibodies affecting the ocular muscles.

Exophthalmos is usually self-limiting and may even regress. Sleeping in the propped up position and lateral tarsorrhaphy may help to

protect the eyes, but have no effect on progression of the condition. Hypothyroidism increases the condition.

Improvement has been reported with high doses of prednisone; its local administration is however, risky especially in the presence of venot congestion. Thyroid ablation has not proved effective, on the contrary it may aggravate a pre-existing exophthalmos which is only checked by postoperative

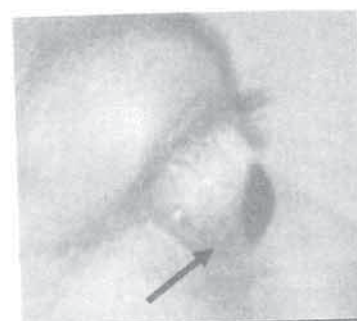


Fig. 26.18. Conjunctival injection with exophthalmos.

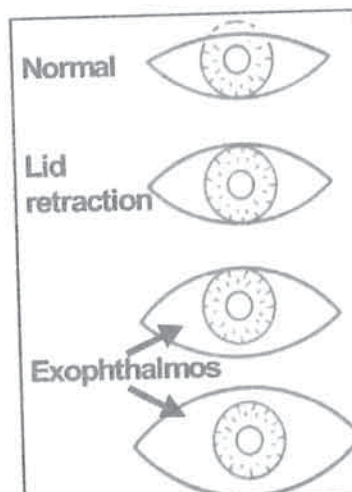


Fig. 26.19. Lid retraction and true exophthalmos.

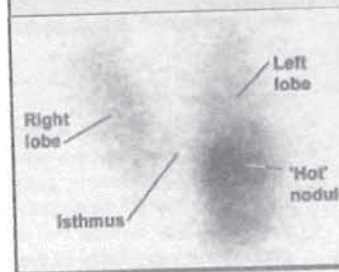


Fig. 26.20. On an isotope scan an autonomous toxic nodule looks hot (dense uptake) while the rest of the gland is suppressed because of the low TSH level.

administration of L-thyroxine. Orbital decompression may be indicated when the eye is endangered. For clinical detection of exophthalmos two methods are available:

1. Naffziger's method. The examiner stands behind the patient, with the head tilted backwards. In true exophthalmos the eyeballs protrude beyond the plane of the superciliary ridge.
 2. Russell Frazer's method. Examining the patient from the side with the eyes lightly closed, will determine the depth of the groove between the orbital margin and the covered globe (shallow in true exophthalmus).
- (b) False exophthalmus is due to retraction of the upper eyelids (Fig. 26.19), and accompanies both diffuse and nodular toxic goitres. It is due to contraction of the levator palpebrae superioris (Muller's muscle) pulling the upper eyelid's tarsus and superior conjunctival fornix upwards. The high level of thyroid hormones sensitizes this unstriated muscle to the effect of circulating catecholamines. False exophthalmos usually disappears when the hyperthyroidism is treated. Beta-adrenergic blocking drugs as guanethidine eye drops improve false exophthalmos.

- Eye signs, other than exophthalmos, include
 - Dalrymple's sign. Appearance of a rim of sclera between the upper eyelid and the cornea.
 - Stellwag's sign. Staring look with infrequent blinking.
 - Von Graefe's sign. The upper eye lid lags behind the moving eyeball as the patient looks down without moving the head.
 - Joffroy's sign. Lack of forehead wrinkling on looking upwards without moving the head.
 - Moebius sign. Defective convergence due to muscle paresis.

10. Local signs of toxic goitre in the neck

- In diffuse toxic goitre, the thyroid gland is diffusely enlarged. It is usually a slight to moderate enlargement. The gland is soft but infrequently firm in consistency. A thrill and bruit may be detected at the upper pole over the superior thyroid arteries. The surface of the gland is smooth and the overlying skin may be warm. The gland may show expansible pulsations.
- In nodular toxic goitre, the thyroid gland is firm in consistency with a nodular surface. The size of the gland may be large.

Diagnosis

Clinical examination provides adequate basis for the diagnosis in most cases.

Thyrotoxicosis should be suspected in

- Children with growth spurt, behavioural problems or myopathy.
- Elderly persons with unexplained tachycardia or arrhythmia (apathetic hyperthyroidism).
- Unexplained diarrhoea.
- Unexplained loss of weight.
- Resistant heart failure (marked thyrotoxicosis).

Table 26.2. Clinical differences between the two main types of thyrotoxicosis		
	Graves' disease	Toxic nodular goitre
Age	Young	Elderly
Onset	Abrupt	Gradual
Course	Exacerbations & remissions	Steady
Nervous symptoms	+++	+
Metabolic manifestations	+++	+++
Cardiovascular manifestations	+	
Eye signs	i-++ exophthalmos	Usually no exophthalmos
Thyroid	Diffuse enlargement soft and vascular	Multiple or solitary nodules

Investigations

- Thyroid function tests T_3 , T_4 , TSH and ^{123}I uptake test confirm the diagnosis.
- T_3 thyrotoxicosis is identified by elevated free T_3 .
- Toxic adenoma shows as a hot nodule on ^{123}I scanning with suppression of the uptake of the surrounding thyroid tissue (Fig. 26.20).

Treatment

The treatment of thyrotoxicosis comprises three options

- Medical treatment.
- Radioactive iodine.
- Surgery.

(A) Medical treatment

Aim is to restore the patient to an euthyroid status, then to prescribe a maintenance dose for a prolonged period in the hope that a permanent remission occurs. A permanent remission is to be expected in patients who have thyroid antibodies at first presentation.

Medications

- Carbimazole.** It blocks iodine binding to tyrosine and decreases antibody titres. It is the most commonly used drug in Egypt. The initial dose is 10 mg three times daily with a maximum dose of 60 mg daily. There is a latent interval of 7-14 days before clinical improvement takes place. When the euthyroid state is reached, the dose is gradually reduced to 5 mg two or three times a day, and is maintained for 12-18 months.
- Propylthiouracil (100 mg t.d.s).** It blocks iodine binding to tyrosine and prevents peripheral conversion of T_4 to the more active T_3 .
- Potassium perchlorate.** It competitively inhibits iodide uptake by the thyroid.
- Propranolol or other beta-adrenergic blockers** which inhibit the cardiogenic effect of thyroid hormones by beta-blockade. Non selective beta-adrenergic blockers e.g. propranolol must not be used in asthmatic patients. In such cases selective beta-blockers as atenolol must be used.
- Iodides.** They reduce the effect of TSH on the thyroid gland and inhibit iodine binding. They also reduce the vascularity of the thyroid gland and lead to storage of colloid within the acini. Their effect is only temporary and so they cannot be used for long term control. Their main use is for preoperative preparation.

Indications

- Primary thyrotoxicosis.
- Mild thyrotoxicosis.
- Children and young patients because surgery may affect their growth.

4. Pre-operative preparation.
5. Post-operative recurrence.
6. Refusal of surgery.
7. Small gland.

Advantages

- Avoiding surgical risks.
- Avoiding the possible hazards of radioactive materials.

Drawbacks

1. There is no way to accurately predict which patient is liable to pass into remission.
2. High relapse rate which amounts to 60% within 2 years from stopping treatment.
3. Further enlargement of the gland may occur.
4. Adverse effects of the drugs Thiouracil and carbimazole may cause gastrointestinal upset, rashes, arthralgia and reversible bone marrow depression leading to agranulocytosis. Blood picture should be performed every two weeks.

Contraindications

1. Toxic nodular goitre. This will ultimately require surgery.
2. Retrosternal toxic goitre. This will ultimately cause respiratory obstruction.

(B) Radioactive iodine

Mechanism of action. Radioactive iodine destroys the thyroid cells thus reducing the mass of functioning thyroid tissue.

Dose. The suggested dose is 160 uCi per 1 gm of thyroid tissue. The response is usually slow, a substantial improvement is anticipated by 8-12 weeks, if not a second dose or several doses may be required, which is the case in 20-30% of these patients.

Possible side effects. Radioactive iodine treatment has long been blamed for causing genetic damage, leukaemia or damage to the foetus if inadvertently administered in early pregnancy as well as increasing the incidence of thyroid carcinoma in adults 10-15 years after its administration. However, no substantial evidence has been definitely established regarding all these adverse effects and so the minimum age for its use in many centers is now 25 years.

Indications

1. Diffuse toxic goitre.
2. Thyrocardiac patients.
3. Refusal of surgery.
4. Recurrence after surgery.

Advantages

- Avoidance of surgery
- Avoidance of prolonged medical therapy.

Disadvantages

Progressive destruction of the gland resulting in thyroid insufficiency in up to 75-80% of cases after 10 years and so an indefinite follow-up is mandatory. Inadequate for the treatment of secondary toxic goitre.

(C) Surgery

The aim of surgery is to remove most of the thyroid gland leaving an equivalent of 4-5 gm of thyroid tissue on each side. Solitary toxic nodule is treated by ipsilateral total

lobectomy. Some surgeons nowadays advocate total thyroidectomy for the treatment of toxic goitre.

The patient should be in a euthyroid status before the operation, otherwise he may develop a post-operative thyroid crisis.

Preparation by carbimazole and propranolol is usually satisfactory. Some surgeons recommend prescribing iodides (Lugol's iodine 10 drops t.d.s.) for 10 days preoperatively to diminish the vascularity of the gland and render it more firm. A sleeping pulse of 80 min is considered as effective preoperative preparation.

Advantage

1. Rapid cure
2. High rate of success.

Indications

1. Secondary toxic goitre.
2. Severe primary thyrotoxicosis.
3. Retrosternal toxic goitre.
4. Failure of medical treatment.
5. Occurrence of side effects due to medical treatment.

Drawbacks

1. Morbidity and mortality. These are negligible in experienced hands.
2. Recurrence rate less than 5%.
3. Thyroid insufficiency at an incidence of 20-45%.
4. Parathyroid insufficiency in less than 0.5% but is a definite risk even in experienced hands because of the high incidence of anatomical variation of the site of the parathyroid glands.

Special problems in management

Thyrotoxicosis with pregnancy

- Radio-iodine is absolutely contraindicated, because destruction of the fetal thyroid would result.
- Antithyroid drugs at conventional doses would result in foetal hypothyroidism with the development of goitre that may obstruct the airway. Minimum doses antithyroid drugs supported by B-blockers, reduce this risk. The dose of an antithyroid drug is adjusted according to the serum level of free T₄.
- Surgery after a short course of antithyroid drugs and propranolol proved to be safe during the second or third trimester.
- During lactation propyl-thiouracil is recommended as it is excreted in a harmless very low concentration in milk.

Thyrotoxicosis in children

- Radioiodine is contraindicated for children.
- Conventional surgery is either followed by a high recurrence rate, due to high activity of the cells in the young or followed by a hypothyroid state which affects later growth.
- It is preferable to tide them over by antithyroid drugs until the late teens.

The thyrocardiac patient

- The cardiac condition takes priority in management.
- Thyroidectomy is ideal after control of the cardiac status.
- If it is not permissible, radioiodine is used followed by antithyroid drugs until the effect of the former appears (6 weeks).

Proptosis of recent onset

- It is not preferable to terminate the toxic status abruptly by surgery or radio-iodine, for fear of the theoretical risk of progressing to malignant exophthalmos.
- Antithyroid drugs are used until the proptosis has been stable for 6 months, after which thyroidectomy is permissible.

Hypothyroidism**Aetiology**

1. Failure of development of the thyroid gland.
2. Dyschromogenesis means congenital deficiency of one of the enzymes necessary for the formation of thyroid hormones, e.g. peroxidase enzyme.
3. Failure of secretion of TSH by the anterior pituitary, e.g. in Sheehan's syndrome.
4. Primary myxoedema is supposed to be an autoimmune disorder. It may be associated with Addison's disease.
5. Iatrogenic following subtotal thyroidectomy or radioactive iodine treatment particularly for toxic goitre.
6. In late stages of Hashimoto's thyroiditis.

Clinical picture

Hypothyroidism in infants or children leads to cretinism while in adults the term myxoedema is applied to severe cases of hypothyroidism.

Cretinism

All the parameters of normal development are impaired. The infant does not grow properly, he is stunted, slow to develop his teeth or to walk. The child has an apathetic look with depressed nose, thick lips and thick protruding tongue. The abdomen is distended (potbelly) and there is usually an umbilical hernia (Fig. 26.21).



Fig. 26.21. Cretinism

Myxoedema

- The symptoms of myxoedema are nearly opposite to those of thyrotoxicosis. The patient complains of lethargy, weakness, loss of appetite, putting on weight and intolerance to cold weather.
- On examination the patient looks apathetic with sluggish reaction. The face is puffy (Fig. 26.22) with supraclavicular pads of fat. The skin and hairs are dry. The hands are cold. There is bradycardia with a low volume pulse. The voice of the patient is coarse. Pericardial effusion, carpal tunnel syndrome, deafness and ataxia may be present.
- Complications. The danger of myxoedema is that the diagnosis may be missed and the condition may progress to coronary thrombosis due to hyper-cholesterolaemia. A myxoedematous patient if subjected to a stressful situation may develop coma.



Fig. 26.22. Myxoedema

Investigations

- T3 and T4 levels are low.
- TSH is markedly raised.

Treatment

Replacement by L. thyroxine 0.2-0.3 mg/day for adults is satisfactory. Very early treatment of cretin infants may save these patients, otherwise the changes are irreversible.

Thyroid neoplasms – Classification

The thyroid gland contains follicular and parafollicular (C) cells. Tumours of the thyroid gland can be classified as follows:

1. Tumours arising from follicular epithelium:

<ul style="list-style-type: none"> ▪ Benign ▪ Malignant 	<ul style="list-style-type: none"> Follicular adenoma Differentiated Undifferentiated 	<ul style="list-style-type: none"> Papillary carcinoma Follicular carcinoma Mixed carcinoma Anaplastic carcinoma
---	--	--
2. Tumours from parafollicular epithelium: Medullary carcinoma.
3. Tumours from lymphoid elements: Malignant lymphoma.
4. Rarely the thyroid gland is infiltrated by metastatic deposits or by local infiltration from a nearby lesion.

Follicular adenoma

- Presents as a solitary nodule.
- The main concern is to differentiate it from follicular carcinoma which may have the same clinical picture. The main differentiating point during histopathological examination is to detect evidence of capsular or vascular invasion in carcinoma. That is why fine needle aspiration cytology is not reliable test in this condition.
- Treatment. Lobectomy of the side of the adenoma plus isthmusectomy.
- Many authors deny now the existence of papillary adenomas and consider them a carcinoma.

Thyroid cancer**Predisposing factors**

1. External irradiation of the head and neck region in children which was previously used for treatment of haemangiomas and thymic gland enlargement.
2. Follicular carcinoma is the commonest type to develop in endemic goitrous area possibly due to continuous stimulation of the thyroid gland by TSH.
3. A genetic element may be present as the lesion is more common in certain families.
4. Malignant lymphoma may occur on top of Hashimoto's disease.

Types

The differences between the three main types of thyroid carcinoma are illustrated Table 26.2.

Table 26.2. Differences between the commonest three types of thyroid carcinoma

	Papillary	Follicular	Anaplastic
Incidence	60%	17%	13%
Age	May occur in children and young adults	Middle age	Elderly
Sex FM	3.5:1	2:1	11:3
Microscopic picture	Papillary projections (Fig. 26.23) formed of connective tissue covered by a single layer of epithelial cells with pale empty nuclei. Laminated calcified bodies (Psammoma bodies) are often present	Thyroid follicles with a variable degree of differentiation, Solid sheets may be present. Diagnosis depends on finding capsular or vascular invasion (Fig. 26.24), or on detecting metastases	May take the form of spindle cell or large cell type
Multiplicity	Up to 80%. May be due to multicentricity or intrathyroid lymphatic spread	Rare	
Spread	Mainly lymphatic. Blood spread may occur with tumours that extend outside the thyroid	Mainly blood spread	Mainly direct invasion. Lymphatic or blood spread.
10-year survival	90%	Encapsulated 97% Invasive 70%	Most patients die within 1-2 years

If the tumour contains both papillary and follicular elements it is managed as papillary.

Medullary carcinoma

- This tumour accounts for about 6% of malignant neoplasms.
- The tumour arises from the parafollicular cells and is, therefore, considered a part of the apudomas. It secretes calcitonin.
- The tumour may occur alone, or it may be associated with hyperparathyroidism and pheochromocytoma constituting (MEN IIa).
- The tumour may be familial and in this case it is more common in children and young adults.
- Microscopically the tumour is composed of sheets of neoplastic cells set in a hyaline stroma which may contain amyloid material. Mitotic rate of growth is usually low.
- It may spread to lymph nodes in 50-60% of cases or gives rise to blood borne metastases.

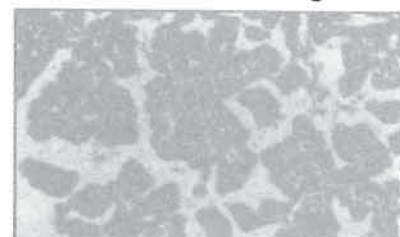


Fig. (26.23). Microscopic picture of papillary carcinoma of the thyroid gland.

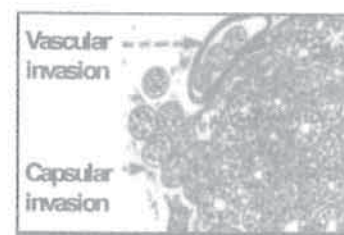


Fig. (26.24). Capsular and vascular invasion are the features that diagnose follicular carcinoma.

Clinical features

- The usual complaint is that of a thyroid swelling which is enlarging rapidly over some months. Sometimes, the swelling is painful and the pain may be referred to the ear (referred along the auricular branch of vagus).
- If the lesion is advanced, the patient may complain of compression manifestations as dyspnoea or change of voice due to involvement of the recurrent laryngeal nerve.
- Examination reveals a lump, firm to hard in consistency, occupying a part or the whole of the thyroid gland. There may be some restricted mobility of the thyroid.

- In anaplastic tumours, the rate of growth will be more rapid and there will be early infiltration of the surrounding structures as the trachea, recurrent laryngeal nerve or the muscles.
- The examiner should look for lymphatic or blood borne metastases, the latter occur usually in the bones particularly the spine, skull or neck of femur, in the lungs or in the brain.
- Some low grade differentiated tumours present exactly as benign lesions. Any solitary nodule of the thyroid particularly in young males is suspicious. Medullary carcinoma may produce diarrhoea in 30% of cases due to production of 5 hydroxytryptamine or prostaglandins.
- Some patients may present, by metastatic lymph nodes in the jugular chain while the primary papillary carcinoma is not palpable. This is called occult carcinoma.

Investigations

1. **Thyroid isotope scanning** is of limited diagnostic significance. Malignant lesions usually appear as cold areas in the scan (Fig. 26.25), but it should be remembered that many benign lesions as cysts or haemorrhage in a cyst or degenerative nodules or thyroiditis appear also as cold areas.
2. **Ultrasound** examination can prove whether the lesion is cystic, solid or cystic with solid projection.
3. **Aspiration of a cyst.**
 - a. A benign cyst is characterized by:
 - i. The aspirate is clear.
 - ii. Complete disappearance of the cyst.
 - iii. No reaccumulation of fluid.
 - iv. Cytological examination is free.
 - b. A cystic malignant lesion is suspected when:
 - i. The aspirate is haemorrhagic.
 - ii. A residual lump is present.
 - iii. Rapid reaccumulation of fluid in the cyst.
 - iv. Cytological examination may be positive for malignant cells.
4. **Fine needle aspiration cytology (FNAC).** The role of this simple, rapid and inexpensive investigation is becoming increasingly reliable. An experienced pathologist can give an accurate result in about 90% of cases. In follicular carcinoma FNAC may not be very conclusive as the diagnosis of this lesion depends upon demonstration of capsular or blood vessel invasion.
5. **Chest x-ray** to detect pulmonary deposits.
6. **Indirect laryngoscopy** to check the mobility of the vocal cords.

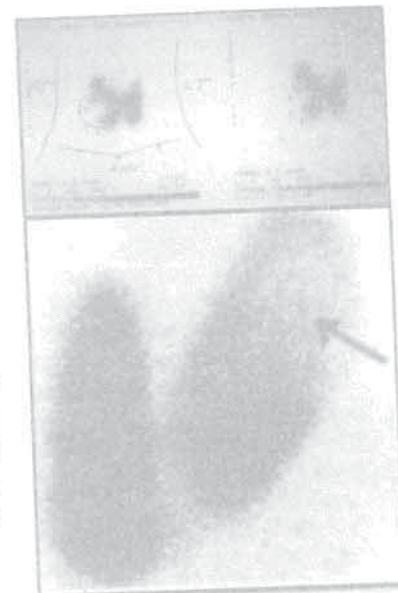


Fig. 26.25. Cold nodules on an a thyroid isotope scan are suspicious of malignancy.

REMEMBER

- The Ps of papillary thyroid cancer**
- Papillary cancer
 - Popular (most common)
 - Psammoma bodies
 - Palpable lymph nodes (in one third of cases)
 - Positive (excellent) Prognosis
- The Fs of follicular cancer**
- Follicular cancer
 - Far-away metastases (blood spread, commonly to bones)
 - Female (2:1 ratio)
 - FNAC not useful
 - Favourable prognosis
- With IV medullary carcinoma remember MEN II**

7. **Tumour markers** Estimation of serum calcitonin is not a routine investigation. It is raised in many patients with medullary carcinoma, and is considered as a tumour marker. Thyroglobulin is a tumour marker for differentiated thyroid carcinoma.
8. If the diagnosis is still in doubt, the best policy is to do surgical exploration of the thyroid and completely resect the affected lobe. It is recommended to do frozen section, so that complete definitive treatment is performed simultaneously.

Treatment

Differentiated carcinoma

Total thyroidectomy is recommended because

1. There is a high incidence of multicentric lesions in papillary carcinoma.
2. Total thyroidectomy ablates all thyroid tissue and makes it possible to detect metastases by scanning and to treat them by radioactive iodine which is not possible in the presence of normal thyroid tissue.

Post-operatively L-thyroxine 0.2 mg/day is prescribed to suppress TSH production as most differentiated carcinomas are TSH dependent.

Management of lymph node metastases

1. If there are no palpable metastatic deposits, only the central lymph nodes are excised.
2. If there are metastases in the posterior triangle, a modified block dissection is performed in which the accessory nerve, internal jugular vein and sternomastoid muscle are preserved.

Management of blood borne metastases

Thyroxine is stopped for a few weeks or substituted by triiodothyronine (to raise TSH) and then iodine scan is performed. If there is evidence of local recurrence or metastases, a therapeutic dose of radioactive iodine is prescribed.

Medullary carcinoma

This tumour does not concentrate radioactive iodine and is not under the control of TSH. Treatment is by total thyroidectomy and resection of metastatic lymph nodes.

Anaplastic carcinoma

- Unfortunately the majority of these patients are inoperable when first seen and are doomed to a fatal outcome within months.
- The usual treatment is external irradiation and chemotherapy.
- If there is tracheal compression, the isthmus is resected.
- Rarely the lesion is operable and an attempt at cure by total thyroidectomy is performed.

Malignant lymphoma

Radiotherapy and chemotherapy.

Prognosis of differentiated thyroid carcinoma

Certain factors increase the risk including

1. Age. Males above 40 and females above 50 years of age.
2. Size of the lesion if more than 5 cm in diameter.
3. Presence of capsular or blood vessel invasion.

Solitary thyroid nodule

Differential diagnosis

1. The nodule may be a part of multinodular goitre, other nodules are not clinically palpable (commonest).
2. Toxic nodule.
3. Colloid nodule.
4. Adenoma.
5. Carcinoma.

Findings that raise suspicion of malignancy in a solitary nodule

1. History of previous irradiation.
2. Young and elderly patients.
3. Recent onset, and rapid growth.
4. Pain.
5. If the nodule is hard, irregular, with limited mobility.
6. Presence of local invasion or lymphatic or blood borne metastases.

Investigations

1. If there is suspicion of toxicity thyroid function tests and thyroid isotope scan are performed. The value of thyroid scan is
 - a. May reveal the presence of multinodular goitre.
 - b. If the nodule is hot, it is toxic and the possibility of malignancy is nearly excluded.
 - c. If the nodule is warm, it is a functioning adenoma and the possibility of malignancy is only 3.5%.
 - d. If the nodule is cold, the possibility of malignancy is 10-16%.
2. Ultrasound can differentiate between a cyst and a solid lesion.
3. Fine needle aspiration cytology (FNAC). This is a simple, fast, inexpensive investigation, which depends upon cytological examination of nodules 0.5 cm or more by an experienced pathologist. It can diagnose most lesions of the thyroid but cannot differentiate follicular adenoma from carcinoma. False positive results occur in 2% while false negative results occur in 3% of cases.
4. True cut needle biopsy It obtains a core of tissue for histological examination of nodules 2 cm or more in diameter. It may cause haematoma.
5. Excision biopsy. The surest method of diagnosis is to do lobectomy (not enucleation) of the affected side, and then perform frozen section histological examination of suspicious cases. Fig. (26.26) is algorithm for the management of a solitary thyroid nodule.

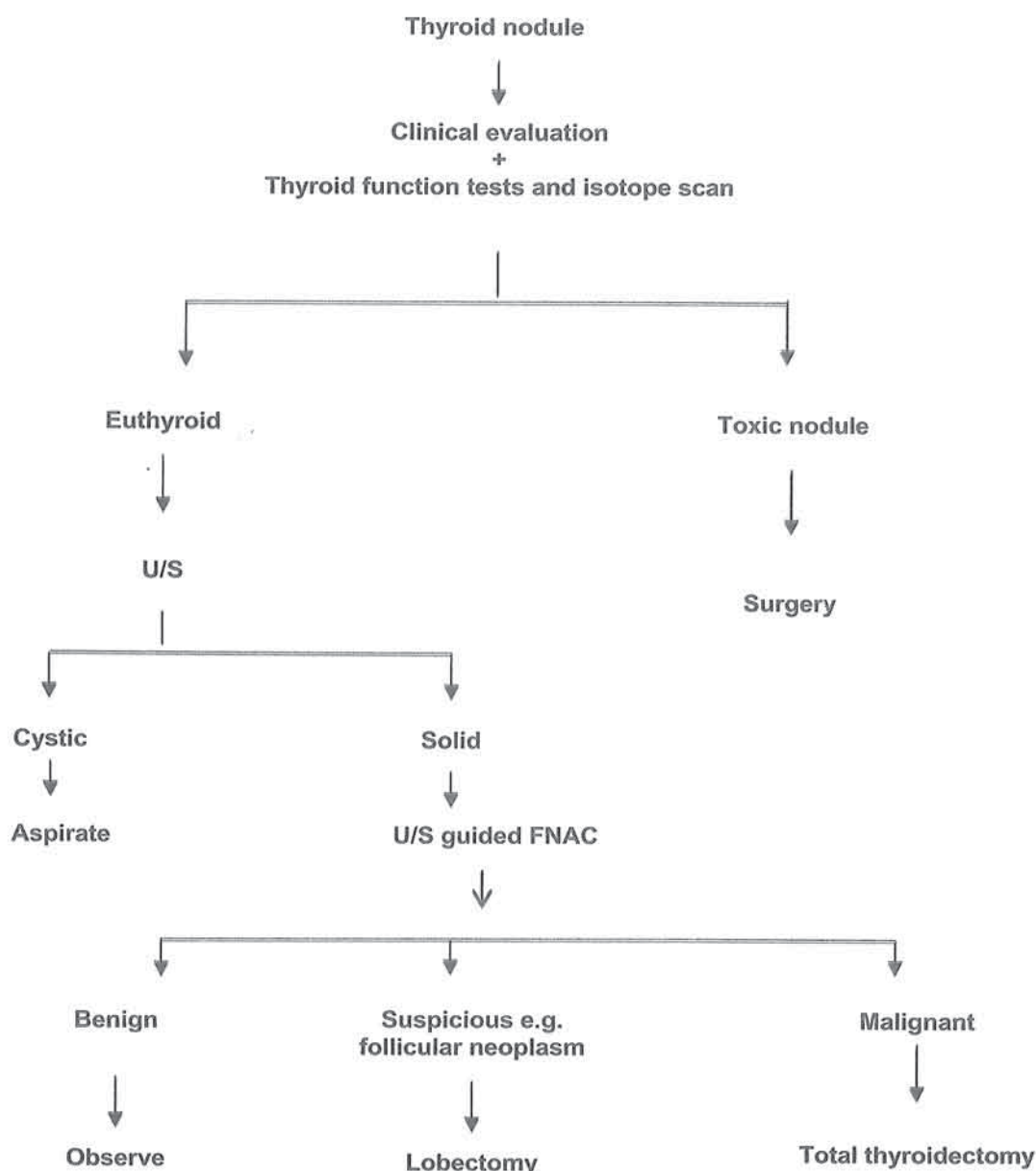


Fig. (26.26) Algorithm for the management of a solitary thyroid nodule

Thyroidectomy operation

Indications

- Simple nodular goiter
 - Diffuse toxic goiter
 - Toxic nodular goiter
- } Subtotal thyroidectomy

Some surgeons nowadays advocate total thyroidectomy for the previous problems

- Solitary nodule → total Lobectomy

- Papillary carcinoma
 - Follicular carcinoma
 - Medullary carcinoma
- } Total thyroidectomy

- Anaplastic carcinoma → thyroidectomy if possible or excision of the isthmus to relieve tracheal compression
- Preoperative
 - If the patient is toxic control the hyperthyroidism.
 - Indirect laryngoscopy to check the mobility of the vocal cords. 3% of people have asymptomatic vocal cord paralysis (medicolegal importance).

Operative procedure

Anaesthetic. general anaesthesia.

Position of the patient. The patient lies supine on the operating table, which is tilted 15° upwards at the head to decrease venous congestion. A sandbag pillow is placed between the shoulder blades with the neck extended (with care particularly in the elderly), thus making the gland more prominent (Fig. 26.27).

Incision. A curvilinear skin crease incision extending laterally as far as the sternomastoid muscle is made about 1 inch above the suprasternal notch (Fig. 26.28). Lower skin incisions have a tendency to develop hyperplastic (keloid) scars.

Raising flaps. The flaps of skin, subcutaneous tissue and platysma are raised upwards to the level of the notch of the thyroid cartilage and downwards to the suprasternal notch.

Strap muscles. The midline raphe between the strap muscles is divided longitudinally. It is preferable to divide the strap muscles for adequate exposure. The pretracheal fascia is opened. A plane of dissection is developed between the strap muscles superficially and the thyroid capsule deeply.

Devascularization. Each thyroid lobe, in turn, is mobilized. Division between ligatures of the middle thyroid vein. The superior thyroid pedicle is ligated within the upper pole to avoid the external laryngeal branch of the superior laryngeal nerve). The inferior thyroid veins are then ligated and divided. The inferior thyroid artery branches are ligated on the surface of the thyroid gland.

The recurrent laryngeal nerve should be identified in its course in the whole operative field. It is sought below the level of the inferior thyroid artery as it passes obliquely upwards and forwards, usually behind the inferior thyroid artery or its branches. The nerve passes into the larynx behind the inferior cornu of the thyroid cartilage.

The parathyroids should be identified before resection of the thyroid gland moreover ligatures near the hilum of the parathyroid as well as excessive use of diathermy should be avoided to limit the risk of devascularization of the gland.

Absolute haemostasis is ensured by ligature of individual vessels or by suturing the capsule of the remnant to the tracheal fascia.

Closure. The pretracheal muscles, if divided, are sutured, as well as the cervical fascia. The wound is closed with suction drainage of the deep cervical space.



Fig. (26.27) Position of the patient for thyroidectomy

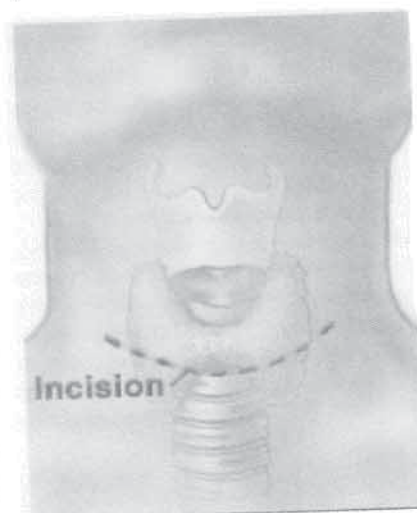


Fig. (26.28) Skin incision for thyroidectomy

Post-operative complications

1. **Haemorrhage.** A tension haematoma in the deep cervical space is usually due to a slipped ligature of the superior thyroid pedicle, but occasionally the bleeding may be from the thyroid remnant or a thyroid vein. Haematoma in this closed space is serious as it may compress the trachea or the carotid vessels. Opening the wound in bed to relieve the tension may be needed before transfer of the patient to the theatre to control the source. A small subcutaneous haematoma may require evacuation or aspiration.
2. **Respiratory obstruction.** The causes of respiratory obstruction following thyroidectomy include:
 - a. Laryngeal edema due to trauma by the endotracheal tube or secondary to the extensive manipulation of the larynx during surgery.
 - b. A tension haematoma in the deep cervical space.
 - c. Bilateral recurrent laryngeal nerve injury.
 - d. Tracheal collapse due to tracheomalacia: The problem of respiratory obstruction is that it may pass unnoticed until the patient develops severe progressive hypoxia and cerebral damage. Whenever, respiratory obstruction is suspected, an endotracheal tube should be immediately inserted and intravenous corticosteroids prescribed.
3. **Recurrent laryngeal nerve paralysis.** This may be unilateral or bilateral, transient or permanent. Permanent paralysis is avoided by proper identification of the nerve during operation. Unilateral paralysis will cause hoarseness of voice and dyspnea on exertion. The voice will usually improve within six months due to compensation by the other vocal cord. Bilateral injury will cause stridor if the abductor fibres are injured (partial injury) or it will lead to aphonia if the whole fibres are damaged (complete injury).
4. **Parathyroid insufficiency.** The incidence should be lower than 0.5%. The condition presents dramatically 2-5 days postoperatively. Delay of the appearance of hypocalcaemia to 2-3 weeks is very rare. Hypoparathyroidism may be due to removal or devascularization of the parathyroid glands. It may be temporary or permanent.
5. **Thyrotoxic crisis (thyroid storm).** This condition represents an acute exacerbation of hyperthyroidism in the form of hyperthermia, tachycardia, irritability and profuse sweating. It follows an operation upon an inadequately prepared patient with thyrotoxicosis, hence it has become very rare. Rarely, it may be the first presentation of undiagnosed thyrotoxicosis in a patient exposed to stress, e.g. infection, or a different surgical procedure.

Treatment

- Intravenous fluids.
- Cooling the patient, e.g. ice packs.
- Oxygen.
- Digoxin for heart failure.
- Sedation.
- Intravenous hydrocortisone.
- Carbimazole 15-20 mg/6 hours.
- Lugol's iodine 10 drops/8 hours or Na- or K-iodide intravenously as a drip.
- Propranolol 40 mg/6 hours orally or 1 mg intravenously as a drip to be repeated if needed.

Post-thyroidectomy dyspnoea

1. **Laryngeal oedema.**
2. **Deep neck haematoma.**
Remove sutures and clot at bedside.
3. **Bilateral recurrent laryngeal nerve injury.**
4. **Tracheal collapse due to tracheomalacia.**

6. **Wound infection and stitch granuloma.** A subcutaneous or deep cervical abscess may need drainage. A granuloma may occur if non-absorbable suture material is used.

7. **Keloid scar.** This is liable to occur if the incision overlies the sternum, and is usually managed by local steroid injections.
8. **Thyroid insufficiency.** This occurs as an insidious process over a period of 2-5 years, at an incidence amounting to 20-45%. Routine estimation of TSH and T₄ should be performed regularly.

Post-operative follow-up

1. Assessment of serum calcium by the 6th week.
2. Follow up every 6-12 month for possible recurrence (5-10%) or development of hypothyroidism. T₃, T₄ and TSH are performed and any necessary treatment prescribed.

Parathyroid glands — basic knowledge

Embryology and Anatomy

Development (Fig. 26.1)

- The parathyroid glands arise from the branchial pouches III and IV.
- The inferior parathyroids are associated with the thymus gland and are derived from the third pouches.
- The superior glands are derived from the fourth pouches.

Number and position (Fig. 26.1, 26.3)

- In 80% of cases there are 4 glands which are symmetrical in position on both sides but in the remaining cases there can be more than 4 asymmetrically placed glands.
- The glands can reach up to the hyoid bone, and down to retrosternal and retroclavicular areas to reach the pericardium.
- The upper glands are fixed in position in 80% of people and are related to the posterior aspect of the upper two thirds of the thyroid lobes.
- The inferior glands are fixed in position only in 60% of cases, as they travel a longer distance, but the commonest site is the area of intersection of inferior thyroid artery and recurrent laryngeal nerve. When these glands enlarge they descend to the mediastinum and become more posterior.

Blood supply

Each gland is supplied by a branch from the inferior thyroid artery that enters at the hilum. Sometimes the superior gland is supplied by the superior thyroid artery.

Histology

- The main cells are chief cells, which are of two types; light and dark; the latter is more active in parathyroid hormone (PTH) production.
- Near puberty oxyphil cells and abundant fat cells appear.
- Hyperplastic and neoplastic glands contain 'water-clear' cells.

Physiology of calcium of homeostasis

- The parathyroid glands secrete parathormone (PTH). Parathormone elevates serum calcium through bone resorption and increased calcium reabsorption in the renal tubules. Together with vitamin D they regulate calcium and phosphorus absorption (Fig. 26.30).
- Vitamin D also increases serum calcium by promoting calcium absorption from the gut.
- On the contrary calcitonin from the parafollicular cells of the thyroid decreases serum calcium through reversing the PTH effect on the bone and kidneys.

- PTH, calcitonin and vitamin D thus work in concert to modulate fluctuations in the level of serum ionized calcium.
- The serum calcium is made of 3 fragments; ionized 50%, protein bound 45% and complexed to organic anions 5%, hence the need for simultaneous assay of plasma proteins especially albumin.
- The factors regulating magnesium metabolism are not well known but PTH definitely plays a role.
- Bones are made of two components; 30% of bone matrix of collagen the major source of hydroxyproline, and 70% inorganic hydroxyapatite. Calcium in bone is divided into two major fractions (1) Labile fraction in physico-chemical equilibrium with the extracellular fluid. (2) Stable fraction bound to organic material; this type is removed only by resorption of bone by osteoclasts.

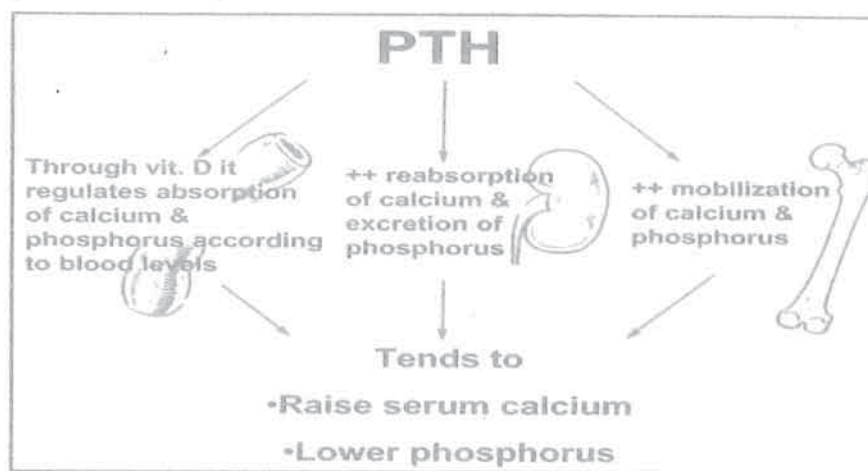


Fig. (26.29) Functions of parathyroid hormone

Hyperparathyroidism

Types

1. Primary hyperparathyroidism. There is autonomous production of the parathormone with loss of the feedback mechanism.
2. Secondary hyperparathyroidism. This type is due to compensatory hypersecretion of the parathormone secondary to low serum calcium.
3. Tertiary hyperparathyroidism. This starts as secondary hyperparathyroidism, but the hyperplastic glands develop an autonomous function.

Primary hyperparathyroidism

Incidence

It is more in females, particularly in the fifth and sixth decades of life. The disease is sometimes familial, and is sometimes a part of the multiple endocrine neoplasia MEN either type I (Werner) or type II (Sipple).

Aetiology

1. A single adenoma [92%].
2. Multiple adenomata [4%].

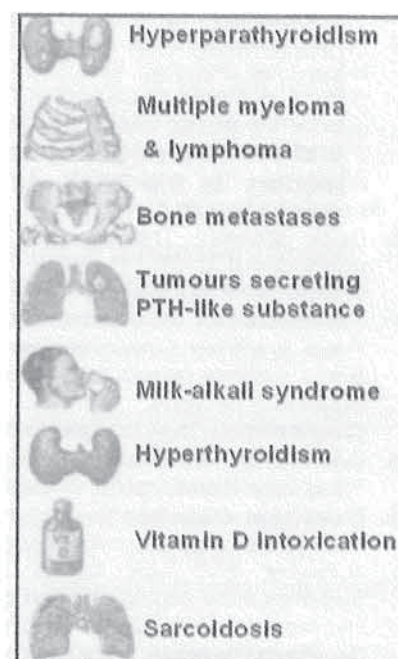


Fig. (26.30) Causes of hypercalcaemia

3. Hyperplasia [3%].
4. Parathyroid carcinoma [1%].
5. Rarely ectopic PTH production by cancers especially of lung, kidney and bladder may produce manifestations of the disease.

Difficulty is encountered in histological differentiation. The presence of a normal rim may suggest adenoma, the presence of locoregional invasion or metastases suggest cancer.

Adenomas and hyperplasia are either chief cell, water clear cell or rarely of the oxyphil cell types.

Clinical presentations

Hyperparathyroidism is more commonly detected due to measurement of serum calcium and phosphate in screening procedures of asymptomatic patients.

In general 80% of patients have renal involvement, and 35% have skeletal involvement.

1. **The earliest complaints** are muscle weakness, anorexia, nausea, constipation, polyuria and polydipsia.
2. **Renal presentations.** These include nephrolithiasis (30-80%), or nephrocalcinosis (5-10%), the latter is irreversible and may lead to renal failure. It is unusual that both lesions occur simultaneously. The patients complain of backache, haematuria, passage of renal calculi of calcium phosphate or oxalate. It is to be mentioned that 75% of patients with primary hyperparathyroidism have calcium stones while only 5% of patients with calcium stones have primary hyperparathyroidism. Hypertension due to renal impairment is responsible for 30% of deaths in patients with persistent hypertension after parathyroidectomy.
3. **Bone disease.** The common skeletal involvements are subperiosteal resorption especially of the phalanges and in severe cases cysts and tufting of terminal phalanges. The skull is the second common site with diffuse granularity and cystic changes. With advanced disease giant cell lesions that look like osteoclastoma (brown tumours) are present. There is severe demineralization of the whole skeleton and multiple pathological fractures may occur. Some patients remain bed ridden for months as the fractures fail to heal. The patient is losing his skeleton in his urine. Serum calcium level is higher with skeletal changes.
4. **Gastrointestinal manifestations.** Peptic ulcer disease is common, while pancreatitis occurs in less than 1% of hyperparathyroidism cases.
5. **Emotional disturbances.** Neurologic and psychiatric problems may occur. The severe forms are not correctable by parathyroidectomy.
6. **Hyperparathyroid crisis** occurs with high serum calcium 16-20 mg per 100 ml, It presents by rapidly developing muscular weakness, nausea, vomiting, weight loss, fatigue and drowsiness.

Hyperparathyroidism is a disease of bones, stones, abdominal groans and psychic moans.

To summarize hyperparathyroidism is a disease of bones, stones, abdominal groans and psychic moans.

Parathyroid carcinoma. Patients are usually symptomatic with skeletal and renal involvements, high elevation of PTH, calcium and alkaline phosphate. The glands are palpable in one third to half of cases.

Differential diagnosis of hypercalcaemia

Causes of hypercalcaemia, other than primary hyperparathyroidism, are.

1. Malignancy (3 groups) characterized by recent onset of hypercalcaemia, elevated sedimentation rate, anaemia, elevated alkaline phosphatase and serum calcium level above 14 mg/l 00 ml.
 - a. Group I (30%). Haematologic malignancies as multiple myeloma and lymphomas.
 - b. Group II (50%). Solid tumours with lytic bone metastases as cancers of breast, lung, kidney and pancreas.
 - c. Group III (20%). Solid tumours without bone metastases. Hypercalcaemia is attributed to their secretion of a humoral factor that binds to PTH receptor in bone (parathormone-like substance).
2. Other causes of hypercalcaemia are milk alkali syndrome, hyperthyroidism, Paget's disease of bone, vitamin D intoxication and sarcoidosis.

Laboratory diagnosis

1. **Serum calcium level.** The normal level is 9-10.5 mg/100ml, values above 13 mg/100 ml are strongly suspicious. The serum calcium level has to be assessed on many occasions, better ionized Ca, analyzed the same day (decreases with refrigeration). Ca and P are simultaneously evaluated together with plasma proteins. 150 mg cortisone for 10 days lower serum Ca only in primary hyperparathyroidism (Dent test). Serum calcium may be normal with hyperparathyroidism in cases of hypoalbuminemia, pancreatitis, vitamin D and magnesium deficiency, and excess phosphate intake. Correction of these disorders may declare hypercalcaemia.
2. **Parathormone immunoassay.** Elevated plasma level of PTH does not establish diagnosis of hyperparathyroidism except with hypercalcaemia.
3. Serum chloride to phosphate ratio above 33 suggests hyperparathyroidism.
4. Increased excretion of calcium in urine.
5. Serum alkaline phosphatase is raised with skeletal lesions.

Localization

The most accurate localization of hyperactive parathyroid glands is by surgical exploration of the neck. In the hands of experienced surgeons nearly 95% of patients with primary hyperparathyroidism are cured at the initial neck exploration.

Helpful preoperative methods of localization include

1. High resolution **ultrasound** with accuracy 76%.
2. **CT scan** has 50% accuracy and is helpful in mediastinal sites.
3. **Technetium** labeled sestamibi scan has proved accurate in the localization of parathyroid adenoma with a sensitivity greater than 80%. Initially both the thyroid and parathyroid glands are visualized, but after a short time the isotope is washed out from the thyroid and the parathyroid is clearly visualized. Combining ultrasound and sestamibi scan has a sensitivity of 95%.

Sestamibi scan has replaced thallium technetium scan.

4. In patients with previous neck exploration, selective venous sampling from certain points along the big veins, e.g. innominate and internal jugular veins may be helpful.

Treatment

Treatment is surgical

- **Parathyroid adenoma.** The adenoma is excised. Operative localization of the adenoma can be helped by giving radioactive technetium and tracing the highest radiation density in the neck by a hand-held probe. The other glands are exposed to ensure that they are of normal size. One of them is biopsied.
- **Parathyroid hyperplasia** is treated by subtotal parathyroidectomy, i.e., excision of three and half glands. Another alternative is total parathyroidectomy with heterotopic autotransplantation of very thin slices in the forearm muscles. Postoperatively PTH serum concentration can be measured from the ipsilateral antecubital vein sample. If patients develop hypercalcaemia, few parathyroid slices can be removed from this easily accessible site.
- **Postoperative care.** Following removal of the parathyroid glands the serum calcium concentration falls to normal in 24-48 hours. Patients with severe skeletal depletion, long standing hyperparathyroidism develop paraesthesia, carpopedal spasm or even seizures. Mild symptoms respond to oral calcium supplementation but severe symptoms need IV calcium possibly with addition of magnesium.

Secondary and tertiary hyperparathyroidism

In secondary hyperparathyroidism there is increased PTH secretion in response to low plasma serum calcium concentration. This occurs in cases of

1. Renal osteodystrophy especially those on haemodialysis associated with elevated serum phosphorus.
2. Malabsorption syndrome
3. Rickets and osteomalacia associated with diminished serum phosphorus.

In tertiary hyperparathyroidism the prolonged parathyroid stimulation causes the chief cell hyperplasia to be autonomous, with elevated serum PTH and calcium.

The treatment of both conditions

Essentially medical by

- Vitamin D.
- Calcium and phosphate binders.

Surgery is indicated for failure to respond to medical treatment by removal of all but about 50 mg of parathyroid or 15 slices 1 mm each. Surgery should be postponed six months after renal transplantation in cases of tertiary hyperparathyroidism as the serum calcium may return to normal after transplantation.

Hypoparathyroidism

Aetiology

The disease is generally uncommon. The commonest causes are;

1. As a complication of thyroidectomy especially for carcinoma or recurrent goitre.
2. Radioactive iodine therapy for Graves' disease.
3. Autoimmune with adrenocortical insufficiency.
4. Following parathyroidectomy operation.
5. Neonatal tetany associated with maternal hyperparathyroidism.

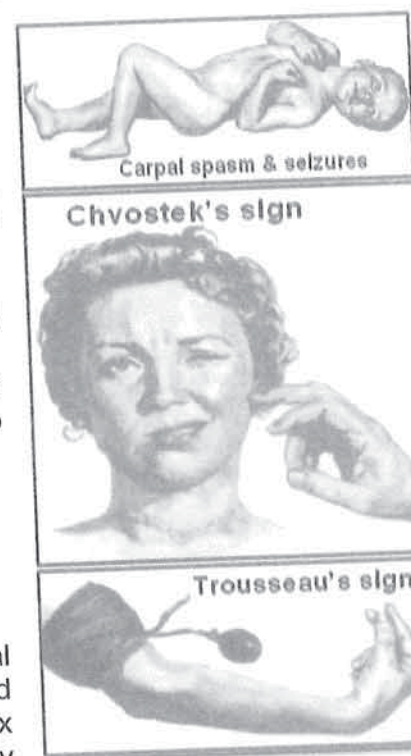


Fig. (26.31): Some signs of hypoparathyroidism

Essentials of diagnosis

- Paraesthesia, muscle cramps, carpopedal spasm, tetany, urinary frequency, depression, and psychoneurosis.
 - Surgical neck scar, positive Chvostek's and Trousseau's signs.
 - Brittle and atrophic nails, defective teeth, cataract, spotty alopecia including loss of eyebrows.
 - Calcification of basal ganglia, cartilage and arteries.
 - Hypocalcaemia and hyperphosphatemia, low or absent serum PTH.
- Some of the signs of hypocalcaemia are shown on Fig. 26.31.

Differential diagnosis

1. Hypocalcaemia may occur after thyroid or parathyroid surgery due to removal, injury or devascularization of the parathyroid glands. After some years the patients may be adapted to low calcium and tetany no longer occurs.
2. Tetany may be also due to alkalosis, hyperventilation, intestinal malabsorption and renal insufficiency.

Treatment

- The aim of treatment is to raise serum calcium, to treat tetany and lower serum phosphate level to prevent metastatic calcification.
- In acute tetany the aim is attained by 10-20 ml of 10% calcium chloride IV and if the response is poor addition of magnesium sulfate 2-4 g/day IV is needed.
- In chronic hypoparathyroidism, that is persistent for more than 3 weeks oral calcium as gluconate, lactate or carbonate is indicated 3 times daily, together with vitamin D. Episodes of hypocalcaemia may occur and need control by dihydrotachysterol. Phosphorus should be limited in the diet by elimination of dairy products. Aluminium hydroxide is prescribed to combine with phosphates and lead to their faecal loss.

Adrenal gland - basic knowledge

Anatomy (Fig. 26.32)

Description

- There are two adrenal glands, one on each side, lying immediately above and medial to the kidneys.
- The adrenal (suprarenal) gland has a canary yellow colour that differentiates it from the surrounding tissues.
- Each gland weighs 4-6g.
- The right gland is in close contact with the inferior vena cava.

Arterial supply

The arterial supply is variable, but usually comes through small arteries from three sources; the phrenic artery, aorta, and renal artery.

Venous drainage

- The venous drainage is more important as it is provided by a constant, single, sizable vein on each side.
- The left adrenal vein joins the left renal vein and is relatively long.

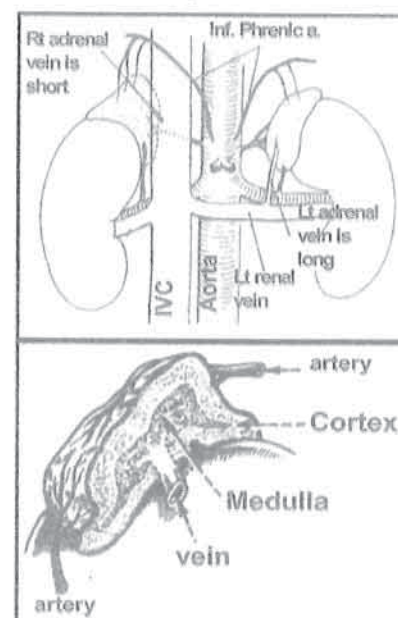


Fig. (26.32) Anatomy of adrenal glands

- The right adrenal vein is very short (2-5mm) and ends in the inferior vena cava. The right vein is, therefore, difficult to ligate and divide, and is easily avulsed from the cava which makes right adrenalectomy more difficult than the left.

Histology and physiology

The gland is formed of two parts; cortex and medulla.

The adrenal cortex, in turn, has three distinct-zones.

From outside in these are

1. **Zona glomerulosa.** The cells are arranged in whorls and secrete the mineralocorticoid aldosterone. The activity of this zone is under control of the renin-angiotensin system.
2. **Zona fasciculata.** The cells are arranged in radial cords and bundles.
3. **Zona reticularis.** The cells are arranged like a network.

The zona fasciculata and reticularis secrete the glucocorticoids cortisol (hydrocortisone) and cortisone as well as androgens and a small amount of oestrogens. The production of glucocorticoids is regulated by a negative feed-back mechanism that is mediated through the anterior pituitary hormone ACTH.

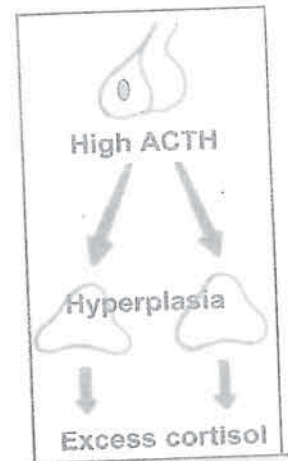


Fig. (26.33) Cushing's disease (pituitary Cushing's syndrome)

The adrenal medulla

- The medulla contains dark chromaffin cells which secrete the catecholamines adrenaline and noradrenaline.
- Embryologically the chromaffin cells are of neuroectodermal origin that are derived from the neural crest. They are, therefore, part of the APUD system, whose tumours (apudomas) are either solitary or form a part of the multiple endocrine neoplasia syndrome.
- Ectopic medullary tissue may be present in the paraganglia, the organ of Zuckerkandl at the origin of the inferior mesenteric artery, in the mediastinum, or in the retrogastric and retropancreatic areas.

Adrenal neoplasms

Tumours of adrenal cortex

These are usually adenomas that arise from the different zones of the cortex. Adenocarcinomas are rare.

1. Non-functioning adenomas.
2. Functioning adenomas produce the following
 - a. Cushing's syndrome.
 - b. Conn's syndrome.
 - c. Adrenogenital syndrome.
 - d. Feminizing syndrome (rare).

Tumours of adrenal medulla

1. Tumours of the sympathetic neurones.
 - a. Benign Ganglioneuroma (chapter 18).
 - b. Malignant Neuroblastoma (chapter 18).
2. Tumours of chromaffin cells Pheochromocytoma.

Cushing's syndrome

Cushing's syndrome is the result of chronic elevation of cortisol production from the adrenal cortex.

Aetiology

There are three distinct causes of this syndrome (Fig. 26.33, 34 & 35).

1. Pituitary Cushing's syndrome (Cushing's disease). An adenoma of the anterior pituitary that produces excess ACTH is the most frequent cause of the syndrome and accounts for about 75% of cases. The high ACTH level produces bilateral adrenal cortical hyperplasia and overproduction of cortisol.
2. Adrenal Cushing's syndrome is the next frequent cause. It is usually the result of a hyperfunctioning adenoma, and rarely a carcinoma of the adrenal cortex.
3. Ectopic ACTH production. An ACTH-like peptide is sometimes produced by a carcinoma of non-pituitary origin. The most likely origin is either the pancreas or the bronchus.

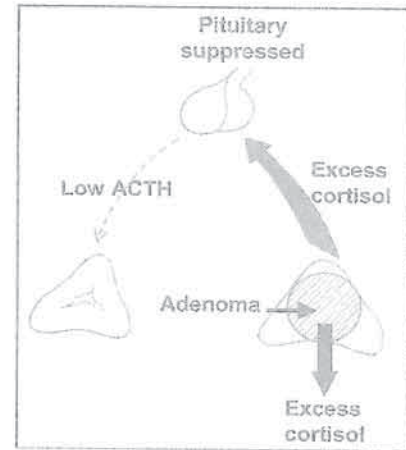


Fig. (26.34) Adrenal Cushing's syndrome

Pathology

A functioning adrenal adenoma inhibits the production of ACTH from the anterior pituitary, with the resulting inhibition of the contralateral adrenal gland.

In Cushing's disease and in cases with ectopic ACTH production both adrenal glands are hyperplastic.

The high cortisol level has the following metabolic consequences

- On carbohydrate metabolism. The hormone is diabetogenic resulting in high blood glucose level.
- On protein metabolism. The catabolic effect of the hormone produces muscle wasting.
- On lipid metabolism. Subcutaneous fat is redistributed in the body. It is deposited in the trunk and the face, leaving slim limbs.
- On water and electrolytes. Cortisol enhances salt and water retention, and excretion of potassium.

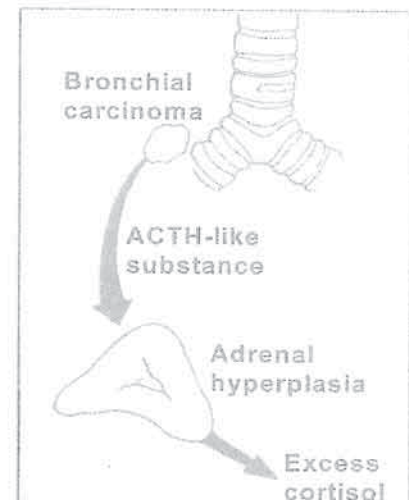


Fig. 26.35 Ectopic ACTH production

Clinical features

Cushing's syndrome is a chronic disease that usually affects young females and, therefore, its manifestations develop over months and years.

1. The change of features often attracts attention to the presence of the syndrome. There is truncal obesity, sometimes with a hump at the back of the neck (buffalo hump), and mooning of the face due to fat deposition. The limbs are slim giving the patient an appearance of "a lemon on match sticks" (Fig. 26.36).
2. Protein catabolism produces muscle weakness, thinning of the skin, abdominal striae and poor wound healing.
3. Diabetes, hypertension, and amenorrhoea.
4. Other features include increased capillary fragility, purpura, osteoporosis, acne, hirsutism, loss of libido, and emotional lability.

Investigations

1. Investigations to prove the presence of Cushing's syndrome.
 - a. Loss of the normal diurnal variations of cortisol level.
 - b. Radioimmunoassay of serum cortisol level.
 - c. Low-dose dexamethazone suppression test. 0.5mg dexamethazone/6h for 2 days is given. In Cushing's syndrome the level of plasma cortisol is not decreased.
 - d. A low ACTH level is diagnostic of an adrenal cause.
 - e. A high level of ACTH is either due to pituitary adenoma or an ectopic origin. The high dose dexamethazone test helps in differentiating. A dose of 8 mg dexamethazone/day is given for 2 days. In pituitary Cushing's cases, the urinary 17-hydrocorticosteroids (end product of cortisol) is reduced, but not in ectopic cases.

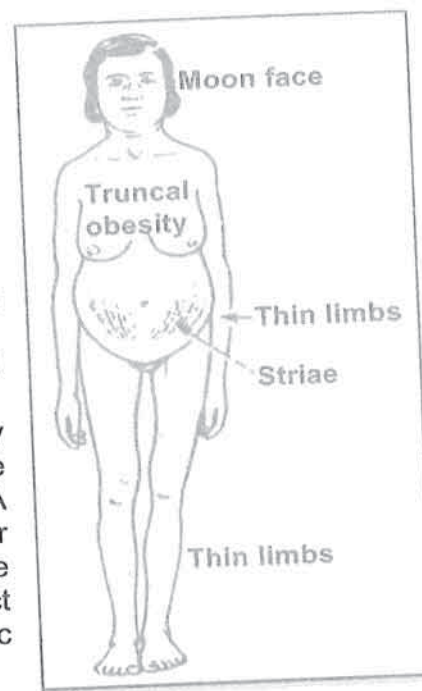


Fig. (26.36) Features of Cushing's syndrome

2. Investigations to localize the lesion include CT scan of the head and abdomen.

Treatment

1. **Pituitary Cushing's syndrome** is best treated by microsurgical excision of the adenoma through the transsphenoidal route. The operation preserves the function of the rest of the gland. If the adenoma is so small to detect, implantation of radioactive yttrium-90 is the alternative treatment. Bilateral adrenalectomy is rarely done nowadays for such cases (Fig. 26.37).
2. Adrenal Cushing's syndrome is treated by adrenalectomy of the affected side. Because of contralateral adrenal gland inhibition the patient needs postoperative administration of cortisol which is tapered gradually with return of adequate function.
3. Ectopic ACTH production is treated by excision of the bronchial or pancreatic carcinoma, whenever possible.

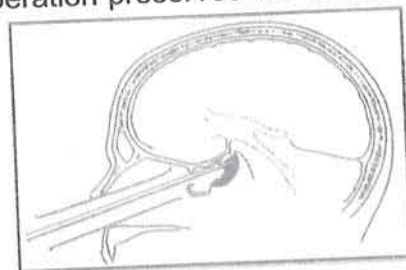


Fig. (26.37) Trans-sphenoidal microsurgical excision of a pituitary adenoma

Hyperaldosteronism

The disease is caused by oversecretion of the hormone aldosterone from the zona glomerulosa of the adrenal cortex.

Aetiology

1. Primary hyperaldosteronism (Conn's syndrome) is caused by the presence of a functioning adrenal cortical adenoma.
2. Secondary hyperaldosteronism occurs with liver, renal, and cardiac diseases. If there is renal ischaemia renin is excessively secreted.

Pathology

Aldosterone produces

- Retention of sodium and water.
- Loss of potassium.

Clinical features

Hypertension is the main feature of the disease. Rarely the syndrome progresses to severe hypokalaemic alkalosis, muscle weakness, tetany, and paraesthesia.

Investigations

The case is suspected in the course of treatment of hypertension.

- A low serum potassium raises the suspicion which is confirmed by finding a high plasma aldosterone.
- CT scan localizes the adenoma.

Treatment

Conns syndrome is treated by surgical excision of the adrenal gland that carries the adenoma. Adrenalectomy should be preceded by control of the blood pressure and hypokalaemia using spironolactone.

Conn's syndrome

Adrenal cortical adenoma.

Hyperaldosteronism.

Sodium retention

The classic clues of Conn's syndrome are

1. Hypertension.
2. Hypokalaemia.

Treatment

- Adrenalectomy of the diseased side
- Preoperative control by spironolactone.

Pheochromocytoma

Pheochromocytoma is a unique neoplasm arising from the adrenal medulla and related chromaffin tissues. The tumours stain deep brownish colour when exposed to chromium salts.

It is one of the causes of surgically correctable hypertension (5-10% of hypertensives), and is encountered in 0.1 to 0.2% of patients with diastolic hypertension. The lesion is commonly undiagnosed. If so it is commonly fatal.

Types

- Sporadic cases usually occur in adults, they are more on the right side, bilateral in 10%, multiple in 10% and malignant in 10% of cases.
- Familial cases occur more in children, and are bilateral in 50% of the patients. The incidence of malignancy, and multiplicity is more than in sporadic cases. They may arise in extra-adrenal sites and may be associated with multiple endocrine neoplasia (MEN II).

Clinical presentations

Pheochromocytoma appears predominantly during the third through the fifth decades of life.

The following presentations are encountered

1. **Hypertension.** This is often paroxysmal but may be sustained or continuously elevated with superimposed crises. There may be cardiac enlargement, anginal pains and postural tachycardia. These manifestations are due to the increased secretion of epinephrine (adrenaline) and/or norepinephrine (noradrenaline). Patients with paroxysmal hypertension are usually more symptomatic than sustained hypertension. Most pheochromocytomas secrete norepinephrine, and some secrete epinephrine as well.

2. Episodes of **headache, palpitation, sweating, and blurring of vision.**
3. Some patients present with **metabolic** symptoms similar to those of diabetes mellitus or hyperthyroidism. These are due to the metabolic effects of epinephrine,
4. Sudden death may occur to some patients due to shock, cardiac irregularities or arrest, or due to cerebral haemorrhage. This fatal presentation may be precipitated by minor trauma or operation.

Complications

- The complications are either due to
1. Catecholamine excess as arrhythmias, diminished blood volume, and cardiomyopathy.
 2. Hypertension as stroke, ventricular strain, myocardial ischaemia and infarction, heart failure, hypertensive retinopathy, retinal detachment and optic atrophy.

Investigations

Laboratory diagnosis

1. Urinary vanillyl mandelic acid (VMA) and metanephrine are useful for screening,
2. When these tests are positive urinary assay for individual catecholamines epinephrine and norepinephrine is indicated. The latter is elevated in 90% of pheochromocytomas. VMA is least specific with false positive results that occur with ingestion of coffee, tea, raw fruits high in vanilla as well as alphas-methyl dopa drugs.
3. Plasma catecholamine assay is useful for the diagnosis of episodic hypersecretion of catecholamines.

Imaging modalities

1. Ultrasound has an accuracy of 80-90%.
2. CT scan (and MRI) can give accuracy up to 100% especially in adrenal pheochromocytoma and can define its relation to the inferior vena cava.
3. ^{113}I methyl iodobenzyl guanidine (MIBG) scintigraphy is very helpful and detect occult metastases.

Indications for screening hypertensives for pheochromocytoma

1. Early onset hypertension (in young).
2. Recent onset with retinopathy or diabetes.
3. Symptomatic hypertension with vasomotor phenomena or diabetes.
4. Episodic hypertension especially with postural hypotension.
5. Pregnancy with vasomotor manifestations as well as hypertension during delivery.

Pheochromocytoma "rule of 10's"

- 10% malignant
- 10% bilateral
- 10% in children
- 10% multiple tumours
- 10% extra-adrenal

Treatment

Treatment is by adrenalectomy of the diseased side.

Preoperative management

Patients with pheochromocytoma have a state of continuous vasoconstriction with diminished blood volume. They are liable to develop severe hypotension once the tumour is removed. To avoid this complication

1. An alpha-adrenergic blocker like dibenzylene is used in increasing doses for 7-10 days preoperatively until orthostatic hypotension occurs. This policy reduced the mortality from 80 to 3%.
2. The same aim can be attained by infusion of large volumes of electrolytes
3. Beta-adrenergic blockers like propranolol are given after alpha blockade for 3-4 days preoperatively to avoid arrhythmias and severe tachycardia.

Operation

Special anaesthetic precautions have to be followed:

1. Premedication should be with pethidine, and scopolamine but not barbiturates.
2. Anaesthesia is best with nitrous oxide and oxygen and not halothane to avoid arrhythmias.
3. Full monitoring is needed during surgery. The following drugs should be available, norepinephrine, phentolamine, lignocaine, propranolol to avoid sudden rise or drop of blood pressure or tachycardia, arrhythmia or cardiac arrest.

Postoperative care

- The patients often needs large amounts of sodium containing fluids in the early post-operative period.
- Hypoglycaemia may occur 3 hours after tumour removal and, therefore, monitoring of blood glucose for the first 5 hours postoperatively is necessary.

Pheochromocytoma symptoms & signs

"Classic triad"

1. Palpitation
2. Headache
3. Episodic sweating

To remember the triad think of the first three letters of the word Pheochromocytoma.

+

Hypertension
Pallor & flushing
Anxiety
Weight loss
Tachycardia
Hyperglycaemia

With Pheochromocytoma



Always look for MEN II

Almost all cases are bilateral

THE BREAST

Surgical anatomy Development

The breast is a modified sweat gland. The mammary glands develop from two ectodermal thickenings, right and left that are known as the 'mammary ridges'. These ridges extend from the axillae to the groins (Fig. 27.1).

In humans only the middle part of the upper third of each ridge persists to form the breast while the rest of the line disappears.

Position and extent

The breasts lie between the skin and the pectoral fascia to which they are loosely attached.

Apparently the adult female breast overlies the area from the second to the sixth ribs, and from the lateral border of the sternum to the anterior axillary line. In anatomical studies, however, a thin rim of glandular tissue of the breast has been observed to extend outside the apparent boundaries of the organ. The actual extent of the breast is important for the surgeon who aims at removal of the whole organ for malignancy. It actually extends

- Upwards to the clavicle.
- Downwards to below the costal margin.
- Medially to the middle line.
- Laterally to the posterior axillary line.

The **axillary tail** (of Spence) is a prolongation of the parenchyma which passes deeply through an opening in the deep fascia to blend with the axillary fat.

Breast components (Fig. 27.2)

The adult female breast has two components

1. **The epithelial elements.** These are responsible for milk secretion and transport. Each breast consists of 15-20 radially-arranged lobes, and each is drained by a lactiferous duct. The ducts converge at the nipple. A lobe is made up of 20-40 lobules, each of which consists of 10-100 alveoli. It is of clinical importance to recognize that the main ducts lie behind the areola, while the lobules occupy the more peripheral part of the breast. The alveoli and ducts are lined by a single layer of epithelium, and the ducts are surrounded by contractile myoepithelial cells which are stimulated by oxytocin and move milk towards the nipple.
2. **The supporting tissue.** Fibrous septa (Cooper's ligaments) extend from the pectoral fascia to the skin, and are responsible for division of the parenchyma

CHAPTER CONTENTS

- Surgical anatomy
- Breast physiology
- Congenital anomalies
- Breast trauma
- Inflammations of the breast
- Fibrocystic disease of the breast
- Cysts of the breast
- Nipple discharge
- Breast neoplasms classification and benign tumours
- Carcinoma of the breast
- Diseases of the male breast



Fig. 27.1. Mammary ridges.

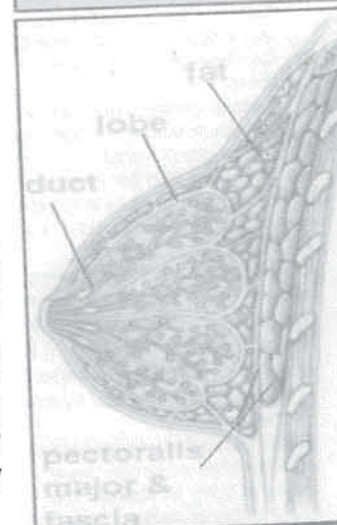


Fig. 27.2. Breast components.

into lobes. The shape of the female breast is due to the fat contained between the fibrous septa. In adolescents and young adults the breast is firm and prominent. With age the glandular and fibrous elements atrophy, the skin stretches, and the breast sags.

Blood supply

Arteries. The arterial supply comes from the following arteries in order of their contribution

1. Internal mammary artery through branches that perforate the intercostal spaces and the pectoralis major muscle. These branches are encountered in the operation of mastectomy, and should be ligated or clamped before their division, otherwise the cut end of the artery retracts and bleeds in the mediastinum and is difficult to control.
2. Lateral thoracic artery, a branch of the axillary.
3. The pectoral branch of the acromiothoracic artery.
4. The intercostal perforators.

Veins. The venous return is primarily through the axillary and internal mammary veins. The intercostal veins are clinically important as they drain into the azygos system and communicate with the valveless vertebral venous plexus. This communication could explain the tendency of breast cancer deposits to affect the axial skeleton.

Lymphatic drainage

This is most important in cases of breast cancer. An extensive lymphatic plexus drains the breast mainly to the axillary and the internal mammary lymph nodes (Fig. 27.3).

Axillary nodes

The axillary lymph nodes constitute the chief draining station of the whole breast, even its medial part. These nodes receive about 75% of breast lymph. There are on average 35 lymph nodes in the axilla that are arranged into

1. Anteromedial (pectoral) group, along the lateral thoracic vessels at the lower border of the pectoralis minor muscle.
2. Posterior (subscapular) group, along the subscapular vessels.
3. Lateral group, along the axillary vein. The posterior and lateral lymph nodes have numerous connections with the anterior chain. Though they do not directly drain the breast, they are potential sites of breast cancer spread through the mentioned connections.
4. Interpectoral (Rotter), a few lymph nodes between the pectoralis major and minor muscles.
5. Central group, embedded in the axillary fat behind pectoralis minor (Level II).
6. Apical group, lying between the pectoralis minor muscle and the clavicle, and situated behind the clavipectoral fascia. The apical group receives lymph from all other groups, and are in continuity with the lower posterior deep cervical nodes that ultimately drain into the subclavian lymph trunk. Lymphatics from the upper lateral part of the breast may drain directly into the apical group (Level III).

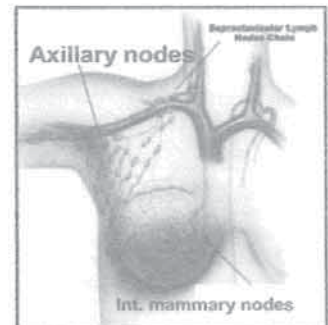


Fig. 27.3: The main lymphatic drainage of the breast is to axillary nodes. Next in importance are the internal mammary nodes.

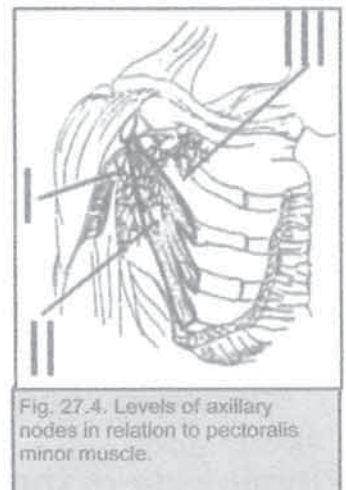


Fig. 27.4: Levels of axillary nodes in relation to pectoralis minor muscle.

For the purpose of determining the prognosis after mastectomy, the axillary lymph nodes are divided by the pectoralis minor muscle into three levels (Fig. 27.4).

- **Level I** nodes are located low in the axilla below the muscle.
- **Level II** nodes are located behind the pectoralis minor muscle.
- **Level III** nodes are located above the muscle in the apex of the axilla.

Within the axilla lymph passes from level I to level II and then level III. The level of lymph node involvement is detected by the pathologist who examines the excised specimen. The prognostic importance of axillary lymph node involvement in breast cancer and their impact on adjuvant therapy is discussed later.

Internal mammary nodes

There are 3 or 4 internal mammary lymph nodes on each side, lying along the internal mammary vessels in the first three intercostal spaces. They receive part of the lymph from the medial half of the breast.

Further lymphatic spread

- Lymph passing through either the axillary or the internal mammary nodes reaches the jugulosubclavian venous confluence. If this is obstructed, then the lymph will pass in a retrograde way to the supraclavicular nodes.
- The posterior intercostal lymph nodes lying along the necks of the ribs have a minor share in breast drainage.
- A few lymphatics pierce the pectoralis major muscle to drain into the occasional lymph nodes that lie between the two pectoral muscles, the interpectoral lymph nodes of Rotter and pass along the intercostal bundles to reach the posterior intercostal lymph nodes.
- Lymph channels from the breast do not cross the midline, but they do cross the diaphragm, where they communicate with liver lymphatics. Lymphatics from the lower inner quadrant may pierce the rectus sheath to reach the peritoneal lymphatic plexus. Previously much emphasis was mentioned about the importance of the subareolar plexus of Sappey and the pectoral plexuses in the lymphatic drainage of the breast. It is now realized that lymphatics of the breast and the skin overlying it pass directly to the corresponding nodes.

Physiology of the breast

Hormonal control

Breast development is under the control of the following hormones.

- Oestrogen, adrenocortical steroids, and growth hormone promote development of ducts.
- Progesterone stimulates the growth of lobules.
- Prolactin is essential for alveolar formation.

Physiological changes

- At puberty. The breast remains dormant until puberty. The onset of cyclical hormonal activity stimulates growth, branching of ducts, and formation of ductules.
- Menstrual changes. During the menstrual years the breast undergoes cyclical changes which can cause heaviness, discomfort, and increasing nodularity during the latter part of menstrual cycle.
- During pregnancy, there is marked lobular development. The high level of ovarian and placental oestrogen, however, inhibits lactation.
- Lactation. Following delivery, reduction of oestrogens increases sensitivity of mammary epithelium to the lactational complex (prolactin, growth hormone, and

cortisol), and milk is formed. Suckling stimulates the release of prolactin and oxytocin. Oxytocin, in turn, stimulates the myoepithelial cells to eject milk into the terminal ducts. These effects are reversed by weaning.

- After menopause the lobules gradually disappear.

Congenital anomalies

Anomalies of the nipple

1. **Athelia.** Absence of a nipple is very rare and is usually associated with absence of the breast (Amazia).
2. **Polythelia.** Supernumerary nipples occur anywhere along either or both the mammary ridges (mammary lines) which extend from the axillae to the groins (Fig. 27.5). An accessory nipple may be mistaken for a mole or a wart,



Fig. 27.5. Bilateral polythelia.

Anomalies of the breast

1. **Amazia.** Absence of the breast is usually unilateral and is often associated with absence of the sternal portion of the pectoralis major.
2. **Polymazia.** Supernumerary breasts are due to persistence of axtramammary portions of the mammary ridge. They may occur in axilla (accessory breast), groin, or even the thigh. They may function during lactation.
3. **Infantile gynaecomastia.** Diffuse enlargement of the male breast which may be unilateral or bilateral. It is caused by the effect of circulating maternal sex hormones. The condition is usually reversible within six months, and, therefore, requires no treatment.

Breast trauma

Blunt trauma to the breast produces two lesions that may be clinically difficult to distinguish from carcinoma, viz, haematoma, and traumatic fat necrosis. Sometimes it is the trauma that draws the patient's attention to an already existing carcinoma of the breast.

Breast haematoma

If there is no external bruising, a deeply seated old haematoma may form a hard mass that greatly resembles a carcinoma. A biopsy will settle the issue.

Traumatic fat necrosis

Blunt breast trauma may cause death of some of the fat cells. The liberated fatty acids combine with calcium to form calcium soaps. The result is one of two forms.

- A cyst that contains thick oily fluid is formed.
- Less frequently a hard mass which resembles a carcinoma is formed. The condition is diagnosed by biopsy. If the lesion is excised, the cut section shows a characteristic chalky white appearance without the yellow specks and gritty texture of carcinoma.

Inflammations of the breast

Acute lactational mastitis and breast abscess

Acute bacterial mastitis is most common during lactation.

Aetiology

Infection is caused by coagulase positive *Staphylococcus aureus*. The organism is claimed to induce clotting of milk in the ducts, producing obstruction and stasis. Organisms from the mouth of the suckling infant gain access through nipple cracks and the openings of the lactiferous ducts. Much less common is the blood-born infection.

Predisposing factors

1. Milk engorgement caused by inspissated milk and epithelial debris that block the ducts.
2. Abrasions to the nipples caused by suckling. A retracted nipple is more likely to be injured by the baby who tries to get hold of it.
3. Poor hygiene.

Pathology

Infection affects any part of the breast and is at first diffuse. The disease usually starts by milk engorgement, which if not properly treated will lead to acute mastitis. *Staphylococci* tend to produce necrosis and pus formation causing a multilocular abscess.

Clinical features

1. **Stage of milk engorgement.** This may affect the whole or a sector of the breast. The patient complains of a dull aching pain. There is persistent pyrexia. Examination reveals enlargement and induration of the breast but there are no physical signs of inflammation.
2. **Stage of acute mastitis.** The pain gets worse and there is continuous higher pyrexia. Examination reveals diffuse swelling, redness, induration and tenderness.
3. **Stage of acute abscess** (Fig. 27.6). Suppuration is diagnosed if
 - Pain is throbbing.
 - Temperature is hectic.
 - Physical signs get localized in the breast.
 - Pitting oedema is elicited in the area of the skin overlying the abscess.
 - Persistence of local signs of inflammation for more than 5 days or of severe systemic upset for more than 2 days after full antibiotic treatment.
 - Breast abscess is one of the sites where the surgeon should not wait for fluctuation to diagnose it.
4. **Stage of chronic breast abscess** (see later).

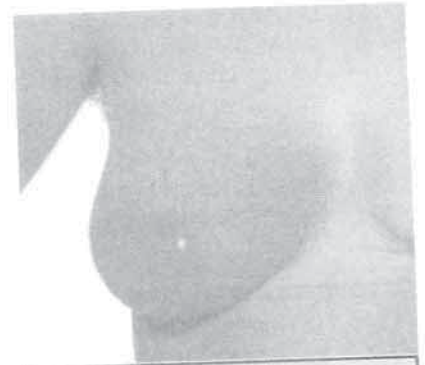


Fig. 27.6. Right breast abscess.



Fig. (27.7) Right breast abscess treated by incision and drainage. The abscess cavity is lightly packed with gauze to promote drainage.

The treatment of pus anywhere in the body is by DRAINAGE.

Treatment

- (A) **Before the development of an abscess** the condition can be medically treated.
- An antibiotic against staphylococci is administered, e.g. a semisynthetic penicillin as flucloxacillin, a cephalosporin, or erythromycin.
 - Support of the breast helps to lessen pain.
 - Local heat.

- The advisability of weaning is controversial, but a reasonable approach is to
 - Advise cessation of breast feeding if the baby has been nursed for more than 9 months. The agents in common use are bromocriptine (parlodel) 2.5 mg twice daily, or stilboesterol 10mg three times a day.
 - If the baby is younger, the patient is asked to use the healthy breast for feeding and to regularly empty the inflamed breast by squeezing and by a breast pump. After resolution of infection the baby can be refeed by both sides.

(B) Abscess formation is treated by incision and drainage (Fig. 27.7).

Under general anaesthesia, a radial or the more cosmetic circumferential incision is made over the most tender area to release the pus which is sampled for culture and sensitivity. The surgeon's finger breaks down the septa between the loculi to form a single cavity. A drain is brought out through the wound, or at the most dependent part of the breast. Antibiotic therapy may be continued for a few days, and the drain is removed when the drainage ceases.

Non lactational mastitis

- The commonest type of non lactational mastitis is that which complicates mammary duct ectasia.
- Inflammation produces a painful, tender, and red periareolar swelling.
- Anaerobes are commonly responsible for the mastitis, and it is, therefore customary to use metronidazole (400 mg four times per day) with flucloxacillin (250 mg four times daily) for five days for its treatment. If an abscess forms, it needs surgical drainage through a small circumareolar incision.
- Spontaneous or surgical drainage of such an abscess may be followed by the development of a mammary fistula at the margin of the areola. These fistulae require surgical excision.

Chronic inflammatory conditions of the breast

Mammary duct ectasia (plasma cell mastitis)

Aetiology is not known.

Pathology

The disease is characterized by dilatation of the major ducts, which fill with pultaceous creamy secretion (Fig. 27.8), and is associated with periductal inflammatory reaction in which round cell infiltration predominates.

Clinical features

Duct ectasia may be asymptomatic, or may present with one of the following

1. Nipple discharge arising from one or more ducts. The discharge may be blood stained, serous, creamy white, or yellow.
2. Retraction of the nipple due to shortening of the ducts.
3. Acute inflammation (mentioned before).
4. Recurrent and chronic inflammation and breast abscess near the areola. Because of the predominance of plasma cells, it is termed 'plasma cell mastitis'. The affected area is hard and may be associated with skin dimpling and nipple retraction simulating a carcinoma.

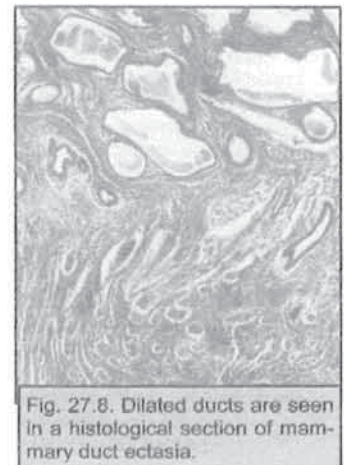


Fig. 27.8. Dilated ducts are seen in a histological section of mammary duct ectasia.

Treatment

Troublesome cases of duct ectasia are treated surgically by excision of the major ducts through a circumareolar incision. Nipple inversion is also corrected.

Chronic pyogenic breast abscess

This is the result of improper treatment of an acute abscess of the breast. Instead of adequate surgical incision, the abscess had been treated by prolonged use of antibiotics or was drained by a small incision. This type of chronic abscess is termed 'antibioma'. The bacteria are killed, yet the pus remains in the breast with excess fibrous tissue formation. The breast becomes chronically thickened and honeycombed with pus. There may be nipple retraction or skin puckering, and so the condition may simulate carcinoma. However, a chronic breast abscess is more painful and is accompanied by a low grade pyrexia. Ultrasound examination and needle aspiration are helpful to differentiate both conditions.

Treatment. A chronic breast abscess is surrounded by a thick fibrous wall and the proper treatment is excision, not only simple incision.

Tuberculosis of the breast

This is a rare disease that is always associated with active pulmonary tuberculosis, or is secondary to tuberculous cervical lymphadenitis. The disease either presents as multiple cold abscesses and sinuses, or as multiple nodules in the breast substance. The axillary nodes are enlarged and matted, and manifestations of tuberculous toxæmia are present. The diagnosis rests on finding the characteristic tuberculous granuloma on histological examination of a biopsy specimen. Treatment is by antituberculous drugs. Mastectomy is reserved for patients with resistant infection.

Fibrocystic disease of the breast

This is the most frequent disorder of the breast. It is also described by other names including mammary dysplasia, ANDI, fibroadenosis, and chronic interstitial mastitis. The last is a misnomer as, in this condition, there is no evidence of inflammation.

Aetiology

- The exact cause is not known.
- The prevalence of the disorder in women aged 30-50 years, and its rarity after the menopause suggests that it is related to ovarian activity.
- Fibrocystic disease is thought to represent a variation or aberration of the normal changes that take place in a healthy breast with the menstrual cycles, pregnancy, lactation, and menopausal involution. The acronym ANDI stands for "Aberrations of Normal Development and Involution".

Pathology

The upper outer quadrant of the breast is the commonest site of affection. The changes which take place include a mixture of the following with a variable proportion

1. **Adenosis** is non-neoplastic glandular hyperplasia, causing multiplication of acini.
2. **Epitheliosis** is solid epithelial hyperplasia within the small ducts. When extensive forms intraductal papillary growths and is termed papillomatosis. In rare cases the is "atypical epithelial hyperplasia" which is accompanied by a higher possibility of developing breast cancer.
3. **Fibrosis** is the replacement of elastic and fatty tissue by fibrous tissue. When fibrosis is extensive it clinically resembles scirrhous carcinoma, and is called "sclerosing adenosis".

4. **Cyst formation.** The cysts may be small (microcysts), or large (macrocyts). They are lined with epithelium and are filled with clear yellow, or sometimes brownish fluid. Cysts are aberrations of involution and are, therefore, most frequent in late premenopausal and menopausal women. Abnormal involution of the supporting connective tissue in the lobules allows the acini to dilate. Obstruction of the ducts by papillomatosis and by the surrounding fibrosis aid in the formation of the cysts.

Clinical features

1. Fibrocystic changes may be completely asymptomatic.
2. The affected lady may present because of an accidentally felt lump. This may be caused by a prominent cyst, aggregation of small cysts, or by the sclerosing adenosis. A palpable thickening may disappear when the patient is re-examined one week after the menstrual cycle. The differentiation from carcinoma is discussed under the differential diagnosis of breast cancer.
3. The most frequent complaint is of multiple, sometimes painful, small lumps that may be unilateral or bilateral (painful nodularity). The lady usually observes this in relation to her cycles.
4. Mastalgia (breast pain) is a common presentation. This is usually a cyclical mastalgia which is an exacerbation of the premenstrual tension. The pain typically occurs few days premenstrually and is accompanied by enlargement and increased nodularity of the breasts.
5. Nipple discharge is usually clear or yellow, but sometimes brown or green.

Investigations

Investigations are usually not required, but the following are indicated to rule out breast cancer in suspected cases.

1. Ultrasound, and mammography show the cysts.
2. A cyst is fully aspirated. It is considered benign if the fluid is not blood-stained, the mass disappears completely after aspiration, and does not recur within two weeks. Cytology of the fluid may be performed to exclude malignancy, but the results are not conclusive.
3. A solid mass is aspirated for cytology.
4. Open biopsy is indicated when experience in interpreting cytology is not available, or if its result is not conclusive. Excised specimens are shown in Fig. 27.9.

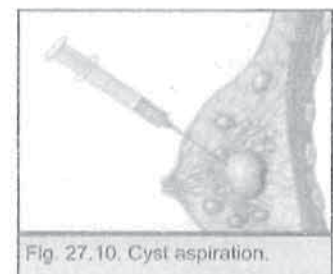


Fig. 27.9. Excised specimens of fibrocystic disease.

Treatment

Treatment is individualized. Exclusion of malignancy and reassurance of the patient are most important.

- Accidentally discovered cases deserve no treatment.
- Cysts are treated by aspiration (Fig. 27.10). A recurring cyst is excised for biopsy.
- Mastalgia:
 - In mild cases reassurance and wearing (night and day) a brassiere that gives good support and protection are usually enough.
 - Giving up caffeine consumption (coffee, tea, and chocolate) may be useful.
 - Prolactin inhibitor as bromocriptine 2.5 mg b.d. gives gratifying results in many patients.



- Danazol, which is a synthetic androgen, is effective in controlling cyclic pain. The dose is 100-200 mg twice daily orally. Its androgenic effects, as acne and hirsutism, limit its use to the unusual severe cases.
- Cases with atypical epithelial hyperplasia, discovered by biopsy, should be instructed to perform a monthly breast self examination. Meanwhile, regular medical follow-up examinations are arranged.

Cysts of the breast

Cysts of the breast usually arise from the duct system (acinar cysts), and occasionally they occur in the stroma (interacinar cysts).

Acinar cysts

1. Retention cysts are due to blockage of the secreting mechanism, either by fibrosis from without or by obstruction from within the lumen. They account for more than 75% of cysts of the breast and are really a manifestation of fibroadenosis (Fig. 27.9). The cyst is often solitary, and varies from one quarter to two inches in diameter. The fluid content may be clear and serous, thick and greenish, or creamy in colour and consistence. Large cysts distended with clear fluid may have a curious blue green colour (blue-domed cysts of Bloodgood). Occasionally, the whole breast is occupied by large cystic spaces.
2. Galactocoele (milk cyst) develops during or shortly after lactation, probably from obstruction to one of the principal ducts. At first, the content is thin and milky and the cyst is tense and thin-walled, but gradually the milk becomes inspissated and the wall thickened and fibrous. The cyst forms a solitary painless swelling situated close to the nipple and dating from lactation. Sometimes a little milky fluid may be expressed from the nipple by pressure on the cyst.
3. Intracystic papilliferous carcinoma.

Interacinar cysts

Cysts arising in the stroma are rare and include

1. Dermoid cysts.
2. Lymph cysts.
3. Blood cysts. These arise either as traumatic haematomas or as other types of cyst with bleeding into them.
4. Sebaceous cysts. These may arise in the skin near the nipple in relation to one of the tubercles of Montgomery.

Treatment

1. Aspiration. About two-thirds of retention cysts are cured by aspiration and provided the diagnosis is certain aspiration should always be tried (see before).
2. Excision. Retention cysts which recur after aspiration and all other cysts should be excised. Superficial cysts are readily excised through a radial incision in the overlying skin.

Nipple discharge

Aetiology

- **Physiological**
 - Milk production with lactation.
 - Serous discharge during pregnancy.
- **Pathological.** The commonest cause is mammary duct ectasia. Table 27.1 illustrates the different causes of nipple discharge.

Table 27.1 Causes of nipple discharge and characters of each

Cause	Nature of discharge
Duct ectasia	Serous, creamy white, yellow, or blood stained. It may be also brown, green or black. It comes out from one or more ducts
Fibrocystic disease	Clear, yellow, brown or green
Duct papilloma	Blood or blood stained discharge from one duct
Duct carcinoma	Rare cause of blood or blood stained discharge from one duct
Contraceptive pills	Serous or milky discharge from multiple ducts
Hyperprolactinaemia	Milky discharge (galactorrhoea) from multiple ducts

Diagnosis

History and examination should provide the following information

1. Nature of discharge.
2. Association with a mass.
3. Unilateral or bilateral.
4. Single duct or multiple duct discharge.
5. The use of contraceptive pills.

Investigations

1. Test for occult blood in the discharge if it is not apparent.
2. Cytology of the discharge for exfoliated cancer cells.
3. Soft tissue mammography and ultrasonography.
4. Duct galactography may be useful in cases of single duct discharge. The test entails cannulation of the duct and injection of a contrast material (lipiodol) prior to taking the radiography. It may show a filling defect or obstruction by a papilloma or carcinoma. The test is not so practical and has been superceeded by the simpler mammography.
5. Serum prolactin estimation in suspected cases of galactorrhoea.

Treatment

1. A palpable mass should be excised for histology and treated accordingly.
2. A single duct bloody discharge calls for excision of this duct by the operation of microdochectomy. A needle with a blunt tip is introduced into the affected duct to act as a guide for the surgeon. This duct, with a rim of the surrounding tissues, is excised and is sent for histological examination.
3. Discharge from multiple ducts with no palpable nor mammographic mass is initially treated conservatively by observation. Rarely the discharge is persistent and troublesome where it is surgically treated by excision of the major ducts.

Breast neoplasms classification and benign tumours

Classification

See table 27.2.

Table 27.2. Classification of breast neoplasms

Benign		Malignant	
Epithelial	Duct papilloma	Epithelial	Carcinoma
Mixed mesenchymal and epithelial	Fibroadenoma	Mesenchymal	Lymphoma (rare) Fibrosarcoma (rare)

Duct papilloma

Pathology

- A duct papilloma is usually situated in one of the main ducts near the nipple (Fig. 27.11) in a young woman.
- Before it becomes big enough to form a palpable lump, it may ulcerate, bleed and cause a blood stained discharge from the nipple.
- If it blocks the duct it may cause a retention cyst.

Clinical features

- The commonest symptom is bloody, or blood stained nipple discharge.
- The accumulation of blood in the duct may be felt as a swelling placed deep, or just lateral, to the areola. The mass is small and fusiform, with its long axis pointing to the nipple. Pressure on the swelling produces the discharge.
- If there is no palpable swelling, pressure on a certain point behind the areola will reveal the discharge coming out from one duct opening.

Investigations

Ductography, done by injecting a contrast material in the affected duct, shows the lesion as a filling defect.

Treatment

Treatment is by excision of the affected duct (microdochectomy) through a circumareolar incision. If there is a lump, it easily leads to the duct. If there is no lump, the duct is identified at operation by passing a blunt tipped needle through the discharging nipple opening. The excised specimen should be histologically examined.

Fibroadenoma

Fibroadenoma is the commonest breast mass of young women. The usual age is between 15 and 30 years.

Pathology

Microscopic picture. A fibroadenoma is a benign neoplasm of the breast which affects both the fibrous and the glandular tissues, but the fibromatous element predominates. Histologically there are two types.

- Pericanalicular (hard) fibroadenomatous are the usual form. These tumours are formed mainly of fibrous tissue that surrounds a few small tubular glands. They tend to be small.
- Intracanalicular (soft) fibroadenomatous contain more glands. They are usually larger and softer than the pericanalicular type.

Gross picture the tumour may be solitary or multiple, firm, with smooth surface. It may be lobulated in big lesions. It is well circumscribed and is never attached to surrounding tissue. The cut surface reveals lobules of whorled white fibrous tissue which bulges out of its capsule.



Fig. 27.12. Mammography of a fibroadenoma.



Clinical features

- Hard fibroadenoma occurs commonly in young women 20-30 years of age while soft fibroadenoma occurs between the ages of 30-50.
- The patient always presents with a painless lump (or lumps) that is accidentally discovered.
- A fibroadenoma is usually small (few centimeters), nontender, spherical, firm, well circumscribed, and with smooth surface that may be bosselated. The characteristic feature, however, is its high mobility within the breast that gained it the name 'breast mouse'.

Investigations

Soft tissue mammography reveals a well-circumscribed lesion (Fig. 27.12).

Treatment

Treatment is by excision (Fig. 27.13), and histological confirmation of the diagnosis.

Cystosarcoma phylloides

- This is a highly cellular type of fibroadenoma that tends to grow rapidly. It was so named by Brodie who used the term phylloides because the cut surface resembles a leaf or fern.
- The term, cystosarcoma, however, is a misnomer as many are not cystic, and it is rarely malignant. It is, therefore, better termed "Phylloides tumour".
- These tumours tend to enlarge slowly and to reach a large size, 20-30 cm in diameter. A big tumour may ulcerate. There are branching like projections of the tumour tissue into the cystic cavities of the neoplasm.
- In spite of its huge size and skin ulceration, the tumour is not attached to the skin.
- Treatment is by wide local excision to prevent recurrence, However, if the tumour infiltrates the whole breast, simple mastectomy is indicated.

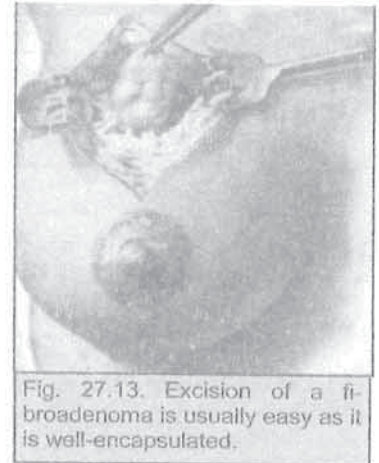


Fig. 27.13. Excision of a fibroadenoma is usually easy as it is well-encapsulated.

Carcinoma of the breast

The breast is the most common site of cancer in women. In the USA one in every nine women is expected to develop breast cancer in lifetime. The probability of its occurrence increases throughout life. The mean age of affection is 60 years. According to the data of the National Cancer Institute of Cairo University, breast cancer in Egypt accounts for about 35% of the total malignancies among Egyptian females. It is, therefore, the commonest malignant neoplasm in Egyptian females.

Aetiology

The exact aetiology of breast cancer is not known. Genetic, endocrinal or environmental factors may have a role.

Genetic factors

- It has been proved that in 5-10% of all cases of breast cancer, there is an autosomal inheritance of a mutant gene. Two genes BRCA1 (on chromosome 17) and BRCA2 (on chromosome 13) are incriminated. These cancers usually occur at a younger age and are multifocal and bilateral.

- Presence of breast cancer in the mother or sister increases the risk by 2.3 times while presence of the disease in both the mother and sister increases the risk by 14 times. 20% of breast cancers are familial.

Endocrinal factors

- The period between the menarche and the onset of the first pregnancy. The longer this period, the more chance of developing breast cancer. So increased risk is present if the menarche occurs below 13 years of age, or if the first pregnancy occurs after the age of 30 years.
- Late menopause after 50 years increases the risk.
- The relation to oral contraceptives or hormone replacement therapy is controversial.

Precancerous lesions

- Moderate or florid epithelial hyperplasia and duct papilloma increase the risk by 1.5-2 times.
- Atypical epithelial hyperplasia increases the risk by 2-5 times.
- Lobular or ductal carcinoma in situ increases the risk by 5-10 times.

Obesity and high intake of saturated fatty acids are associated with an increased risk. Steroid hormones are converted to oestradiol in fatty tissues.

Previous affection with breast cancer. Patients with breast cancer in one side, have an increased risk to develop cancer in the other breast.

Pathology

Microscopic picture

Carcinoma of the breast may arise from the lobules, the ducts or the nipple, with the tumour arising from the terminal duct lobular unit in the majority of cases. The carcinoma may remain within the epithelium (in situ) or, more frequently, it invades (infiltrates) the basement membrane. Accordingly the following histological types are seen

▪ Carcinoma of the ducts

- Non-infiltrating duct carcinoma (ductal carcinoma in situ).
- Infiltrating ductal carcinoma (75%) is shown in Fig. 27.14. All grades of differentiation from anaplasia to well-differentiated tumours may occur.
- Invasive duct carcinoma is the commonest type of breast cancer, 75% (it not otherwise specified, NOS).

▪ Carcinoma of the lobules

- Non-infiltrating lobular carcinoma (lobular carcinoma in situ) is frequently multicentric.
- Infiltrating lobular carcinoma. The lesion is bilateral in 25% of cases.

- **Paget's disease of the nipple.** It is essentially an intraduct carcinoma which begins in the epithelium of a main collecting lactiferous duct and spreads within the epithelium up to the skin of the nipple and down into the breast substance. Histologically there are three characteristic features which are Paget's cells, hyperplasia of all layers of the epidermis, and round-cell infiltration of the dermis. Paget's cells are cl

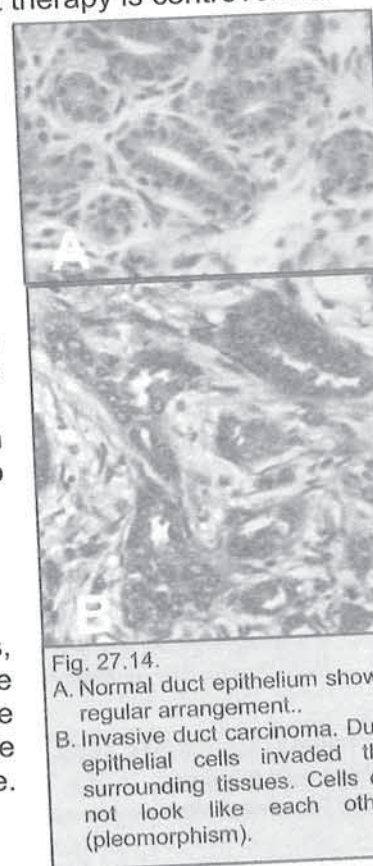


Fig. 27.14.
A. Normal duct epithelium shows regular arrangement.
B. Invasive duct carcinoma. Ductal epithelial cells invaded the surrounding tissues. Cells do not look like each other (pleomorphism).

vacuolated cells with small dark-staining nuclei which occur alone or in clusters in the deeper layers of the epidermis (Fig. 27.15).

Gross types

The commonest site is the upper outer quadrant of the breast.

- **Schirrhous carcinoma** (schirrhous = hard). This is the commonest type (about 75%). The tumour is hard. The cut surface is concave, rough, gritty, and pale grey (Fig. 27.16) with prominent yellow and white flecks (the cut surface of a benign lesion bulges out to become convex, is white rather than grey, and feels smooth and rubbery, not gritty). The gritty sensation is similar to that felt when cutting through an unripe pear. Rapid lymphatic spread occurs through the breast and there may be multiple satellite masses within the breast.
- **Encephaloid carcinoma** (encephaloid = brain-like). The tumour is large, soft, and brain-like. It contains less fibrous tissue than the previous type.
- **Inflammatory carcinoma** (mastitis carcinomatosa) is a rare type, but is the most malignant. It is a fulminant form of infiltrating duct carcinoma that resembles mastitis.
- **Paget's disease** of the nipple is rare. The disease is characterized by nipple erosion. In early stages a mass may not be palpable. A mass may only appear after 2 years from the start of the disease.

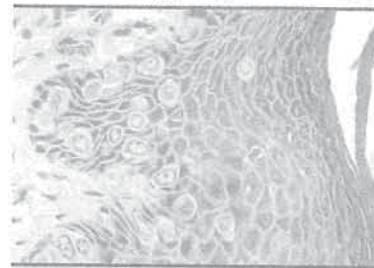


Fig. 27.15. Histological picture of Paget's disease.



Fig. 27.16. Schirrhous carcinoma with nipple retraction

Spread

- **Local spread** can occur through the breast substance, overlying skin, underlying pectoralis major and serratus anterior muscles, and the chest wall.
- **Lymphatic spread** (by embolism and permeation) is mostly to the axillary nodes, next common is the internal mammary chain. Involvement of the supraclavicular nodes is considered an advanced disease. By the time the lymph nodes are histologically affected by malignancy, one should assume that distant organ micrometastases are likely to be present, and the patient is treated accordingly. Lymph node affection is, therefore, an important prognostic factor. Obstruction of the dermal lymphatics by malignant cells produces breast skin oedema that is marked in the dependent part, i.e., the lower half of the breast. The oedematous skin is pitted at the sites of hair follicles, sebaceous glands, and sweat glands giving the appearance of an orange peel, hence the French term "peau d'orange".
- **Blood stream spread** produces metastases in the lungs, bones, brain and the liver. The bones commonly involved by metastases include the lumbar vertebrae, ribs, femur and the skull. The tendency of breast cancer to metastasize to the spine is explained by the free communication of the posterior intercostal veins with the paravertebral plexus of veins.

Previously it was thought that breast cancer spreads locally at first, then by lymphatics, and lastly by the blood stream. This view is no longer accepted, and it is now well realized that carcinoma of the breast may spread by the blood stream very early, producing micrometastases in distant organs (Chapter 10, Fig. 10.12).

Hormone receptors

- About 60% of breast cancers have receptors for **oestrogen** and are termed ER-positive. These tumours get more active under the influence of this hormone. The tumour can be induced to regress if the patient is deprived of oestrogen, or the hormone receptor is blocked by an anti-oestrogen.
- Tumours may also have **progesterone** receptors.
- About 20% of breast cancers exhibit **Her2/neu** receptors which are members of epidermal growth factor receptors involved in cell growth regulation. Overexpression of Her2/neu indicates a worse prognosis.

Clinical features

Symptoms

1. The patient accidentally notices a painless lump in the breast when washing or looking into a mirror. An accidental trauma to the breast may attract the attention of the patient to the presence of the lump.
2. Much less frequently the disease is discovered because of mild breast pricking pain, recent nipple retraction or blood stained nipple discharge.
3. Occasionally breast cancer announces its presence by the appearance of metastases. The latter include an axillary lump, backache caused by spine metastasis, pathological fracture, dyspnoea and pleuritic pain caused by pulmonary metastasis, jaundice caused by liver secondaries, or mental changes and fits due to cerebral deposits.
4. In some Western countries the disease may be discovered by routine screening mammography that is done for the high risk asymptomatic women.

Signs

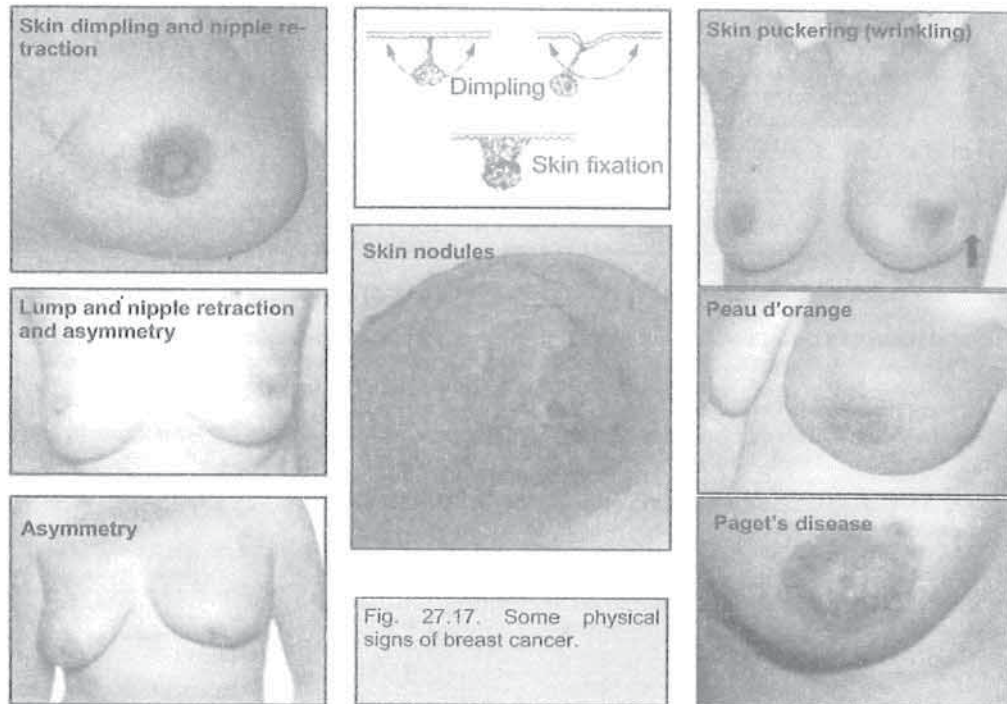
For breast examination, the top half of the trunk is exposed. Both breasts, axillae, arms, and supraclavicular regions are examined.

Some of the following signs (Fig. 27.17) may be detected

- **Breast** may show asymmetry, enlargement, skin dimpling, or skin puckering. Dimpling and puckering of the skin are evident when the patient is sitting and elevates her arms, Peau d'orange, skin nodules and ulceration indicate a locally advanced disease
- **Mass**
 - Hard, irregular and ill-defined.
 - Restricted mobility within the breast substance.
 - Fixation to the skin, underlying muscles, or chest wall, if present, diagnostic of carcinoma. Skin tethering is caused by invasion and shortening of a ligament of Cooper, where the mass can be moved within a certain range without dimpling the skin, but outside this range the skin is dimpled. Skin fixation, on the other hand, is caused by direct infiltration of the skin with the tumour, and the mass cannot be moved at all without moving the skin.
- **Nipple**
 - Recent retraction.
 - Change of direction.
- **Axillary and supraclavicular nodes** Number and mobility of palpable nodes are assessed. It should be noticed that clinical evaluation of the axilla is inaccurate. Impalpable axillary nodes may turn out to have tumour deposits when examined histologically after the operation, and vice versa.

▪ **Distant metastases:**

- Chest examination.
- Hepatomegaly.
- Ascites.
- Pelvic examination for hard deposits or Krukenberg tumour.



Special clinical forms

Pagets disease of the nipple. This is a rare form that constitutes about 1% of breast cancers. The first symptom is often an abnormal pricking sensation of the nipple, with superficial erosion. A tumour mass may not be palpable. The lesion is commonly mistaken for eczema. The diagnosis is established by biopsy of the erosion. Table 27.3 illustrates the clinical differences between Paget's disease and eczema of the nipple.

Table 27.3. Clinical differences between Paget's disease of nipple and eczema

Paget's disease	Eczema
Unilateral	Commonly bilateral
Usually occurs at menopause	Commonly occurs at lactation
No itching	Itching
No vesicles-not oozing (dry)	Vesicles-oozing
Nipple is eroded	Intact nipple
Well defined	Ill defined
A breast lump may be felt	No lump
No response to eczema treatment	Responds to treatment
Starts in the nipple	Starts in the areola

Inflammatory carcinoma. This is a rare aggressive form of breast cancer, which usually occurs during pregnancy or lactation. There is a rapidly growing, sometimes painful, breast swelling. The overlying skin becomes red, oedematous, and warm. Often there is no distinct mass, since the tumour infiltrates the breast diffusely. The picture clinically resembles acute mastitis. The prognosis is poor as it is usually advanced at the

time of diagnosis. Table 27.4. illustrates the clinical differences between inflammatory carcinoma and acute mastitis.

Table 27.4. Clinical differences between inflammatory breast cancer and acute bacterial mastitis

Inflammatory carcinoma	Acute bacterial mastitis
Gradual onset with no or low grade fever	Acute onset with high fever
Progress is slower	Rapid progress
Involves more than one third of the breast	One breast sector is affected
Skin is dusky red	Skin is rosy or fiery red
Mildly tender or non-tender lesion	Markedly tender lesion
Non-tender axillary nodes	Tender axillary nodes
No response to antibiotics in one week is an indication for biopsy	The lesion is either cured by antibiotics or forms an abscess

Carcinoma in situ. In-situ breast cancer is now more frequently recognized due to the use of mammographic screening. 20% of cancers detected by screening are in situ cancers. Two types are recognized (Table 27.5).

Table 27.5. Differences between duct carcinoma in situ and lobular carcinoma in situ

	Duct carcinoma in situ (DCIS)	Lobular carcinoma in situ (LCIS)
Frequency	More common	Less common
Bilaterality and multicentricity	Rare	Common
Microcalcification	Present	Absent
Early detection	Possible	Less likely
Potential for invasive cancer	30-50%	It is a marker of increased risk of malignancy in the same or other breast
Treatment	As invasive cancer	Strict follow up

Differential diagnosis

- **Cases presenting by a lump (mass).** The clinical features of common breast lumps are shown in Table 27.6. The above lesions constitute 95% of breast lumps.

Table 27.6. Clinical features of common breast lumps

	Carcinoma	Solitary cyst	Fibrocystic disease	Fibroadenoma
Age	Usually >35 y	35-55 y	35-55 y	15-30 y
Pain	Painless	Occasionally painful	Occasionally painful	Painless
Surface	Irregular	Smooth surface	Indistinct surface	Smooth, lobulate if large
Consistency	Usually hard	Fluctuation is difficult to elicit, so it feels soft to hard	Firm, ill defined areas of thickening	Firm Highly mobile
Lymph nodes	Probably axillary node enlargement	Free axilla	Free axilla	Free axilla

- **Paget's disease** should be differentiated from eczema of the nipple.
- **Inflammatory carcinoma** should be differentiated from an acute breast abscess.
- **Nipple retraction.** Two types of nipple retraction should be identified.

- Longstanding nipple retraction that dates back to puberty is an innocent finding. It is due to failure of the nipple to follow the spurt of growth of the rest of the breast. The retracted nipple poses difficulty only when the lady is lactating, and is a predisposing factor for the development of mastitis. The problem is spontaneously corrected during pregnancy or lactation.
- Recent nipple retraction in womanhood should be considered seriously. One cause is carcinoma, and the other is mammary duct ectasia. In the latter, the presence of a periareolar mass, as well as the retracted nipple, make clinical differentiation from carcinoma a very difficult task. Other rare causes include chronic non-specific breast abscess and TB.

Staging

The management and prognosis of breast cancer depend on the stage of the disease. Initial treatment is based on the clinical staging, while further management is based on the pathological staging which may differ from the clinical one, e.g. lymph nodes which were clinically impalpable may be found to be histologically involved by the disease.

The two common systems of staging are

A. International TNM staging

T = Tumour

T_{is} Carcinoma in situ. Paget's disease with no palpable tumour.

T₀ No evidence of primary tumour.

T₁ 2cm diameter or less.

T₂ 2-5 cm diameter.

T₃ Tumour larger than 5cm.

T₄ Any size with direct extension to chest wall or to skin; or inflammatory carcinoma

N = Nodes

N₀ No palpable axillary nodes.

N₁ Mobile palpable homolateral axillary nodes.

N₂ Fixed homolateral axillary nodes.

N₃ Palpable homolateral supraclavicular nodes. Oedema of the arm.

M = Metastases

M₀ No evidence of distant metastases.

M₁ Distant metastases.

(B) **Staging of the UICC** (union international contre cancer) is now internationally approved (Table 27.7). It is based on the TNM staging

Table (27.7): UICC staging

Stage UICC	Description	Category	5 year survival (%)
I	T1, N0, M0	Early breast cancer	93
II	IIA T2, N1, M0	Early breast cancer	72
	IIB T3, N0, M0		
III	IIIA T1-3, N0-2, M0	Locally advanced breast cancer	41
	IIIB T4, any N, M0		
IV	Any T, any N, M1	Metastatic	18

Investigations

Aim of Investigations

1. Diagnosis of carcinoma by imaging (mammography, ultrasonography) and biopsy.
2. Staging the disease is routinely done by chest X-ray, ultrasound examination of the abdomen and pelvis, and by alkaline phosphatase (a high level indicates bone or liver deposits).
3. Further tests are needed in special situations. Bony pains require X-rays and isotope bone scan. Suspicion of cerebral secondaries is an indication for CT examination. Whole body PET CT scan can detect metastases throughout the body.

Mammography

This is soft tissue radiology of the breast. In expert hands it is 95% accurate in diagnosing breast cancer. If combined with Tru-cut biopsy, or fine needle aspiration, their accuracy of diagnosis is improved. It is also useful for detecting multifocal lesions in the same or other side, before embarking on radical surgery. A cancer appears as

- A dense opacity that has an indefinite outline from which irregular spicules penetrate into the surrounding breast (Fig. 27.18).
- Clustered microcalcifications (Fig. 27.19).

Mammography is of less diagnostic value in young women, in whom the density of the lesion differs little from that of the surrounding tissue. Digital mammography allows magnification and transfer of images for a second opinion.

Ultrasonography

It can differentiate cystic from solid lesions. A speculated hypoechoic mass more deep than wide suggests a malignant lesion. The addition of colour coded Duplex ultrasound identifies the vascularity of the lesion. Malignant lesions receive blood flow from all around with turbulent speed whereas benign lesions receive blood flow from one side at a low speed. It is particularly useful in young women in whom mammography may not be very helpful. U/S guided biopsy can be done.

Magnetic resonance imaging (MRI)

It is indicated with contrast in certain situations e.g.

- Postoperative scarring to differentiate between fibrosis and local recurrence of malignancy.
- After neoadjuvant therapy to monitor response.
- In the presence of breast implants.

Biopsy

A pathological evidence of malignancy is the corner stone of diagnosis. T different types of biopsy are:

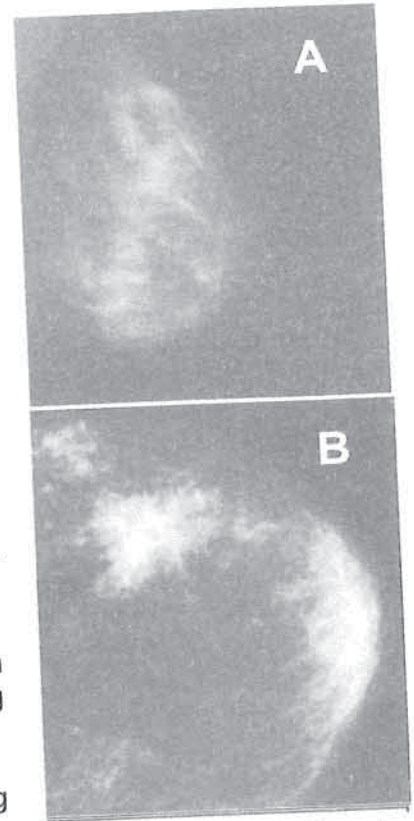


Fig. 27.18. A. Normal mammogram. B. Dense irregular opacity of breast cancer



Fig. 27.19. Clustered microcalcification is characteristic of early breast cancer

1. **Fine needle aspiration cytology (FNAC).** Depends on examination of cells to detect criteria of malignancy in them. The aspiration can be done in the outpatient clinic using a 10-20 ml syringe and a 21-gauge needle. The mass is fixed between the fingers of one hand, and the needle is passed in it several times while keeping constant suction, by the other hand. The suction is then released and the needle is withdrawn out of the breast. This procedure will draw some cells from the lesion into the needle, the contents of which are expressed on a slide and smears are prepared for staining and examination. A skilled cytologist is needed for accurate interpretation.

Sometimes aspiration reveals a cyst. A breast cyst is considered benign if the fluid is not blood stained, the cyst disappears completely with aspiration, and does not recur within two weeks. In case of doubt, the fluid is subjected to cytological examination. Large pleomorphic nuclei, ill-defined cytoplasm and abnormal nuclear chromatin ratio are criteria of malignancy.

FNAC is a very simple, inexpensive, and accurate procedure. It can give a definite diagnosis in 90% of cases. It allows for proper preoperative planning of the definitive treatment. False negative results may occur in up to 10% of cases.
2. **Tru-cut biopsy** is done under local anaesthesia with a special needle that cuts a core of tissue out of the tumour. The obtained specimen allows for histological examination.
3. **Excision biopsy.** The whole mass is excised through a circumareolar or a transverse incision. Paraffin sections are stained and examined to produce a result within two days. Excision biopsy is the most reliable and provides a big enough specimen to allow for hormone receptor estimation as well. It is, however, gradually losing popularity in favour of the simpler fine needle aspiration cytology and Tru-cut biopsy.
4. **Frozen section biopsy.** This is a form of preparation of the specimen for examination by the pathologist. The tumour is either excised or, if large, a small piece is obtained by incising it (incision biopsy). The biopsy specimen is frozen and slides are prepared from the frozen block. A diagnosis is obtained within 20 minutes. Meanwhile, the patient is kept under general anaesthesia, and the surgeon proceeds to obtain haemostasis and close the wound. If the result of examination is negative for malignancy, the patient is awakened. If it is positive, the surgeon proceeds with radical surgery. The possibilities should have been discussed with the patient before the operation, and a consent for mastectomy should have been obtained. Sometimes a firm diagnosis cannot be obtained by frozen section examination, and the result of a paraffin section study should be waited for.
5. **Biopsy from impalpable breast masses.** The radiologist can place a wire inside the lesion under mammographic guidance. At operation, the mass is removed with the wire for histological assessment.

Triple assessment

This means comparing the results of:

- Clinical examination.
- Soft tissue mammography or ultrasonography.
- Fine needle aspiration cytology.
 - o If the three parameters are concordant, the surgeon can rely on the diagnosis.
 - o If the three parameters are not concordant, a further investigation e.g. excision biopsy is needed.

Early detection

This aims at the detection of breast cancer very early in the asymptomatic females in order to offer them a better chance of cure. There are two main methods for early detection of breast cancer.

Breast self examination (BSE)

All women over age 20 should be advised to examine their breasts monthly, one week after the menstrual period. The physician instructs the women as how to conduct a systematic inspection and palpation. If the woman suspects the presence of a lump, skin dimpling, or recent nipple retraction, she should report to the surgeon.

Screening programs

In some Western countries high risk women are subjected to regular clinical examination and mammography. The frequency of examination is every one, two, or three years, depending on the program. Polymorphic (variable in size and shape) dots of calcification detected on mammograms are suspicious and are, thus, subjected to biopsy. Screening programs proved to be most useful in reducing breast cancer mortality in women above 50 years.

Screening soft tissue mammography can detect breast cancer at an earlier stage. This will allow more conservative surgery and improved survival.

Sentinel lymph nodes biopsy

The sentinel lymph node is the first lymph node in the axilla to be affected by metastases. Injection of patent blue violet or a radioactive sulphur colloid near the tumour will allow, identification, excision and immediate pathological examination of the sentinel lymph node.

If the node is positive for metastases → axillary clearance

If the node is negative for metastases → no further excision of lymph nodes.

Treatment

There is still a lot of controversy regarding the ideal treatment of carcinoma of the breast. A multidisciplinary approach involving the surgeon, radiotherapist, and oncologist allows for planning the best treatment for each case.

Treatment depends on the stage of disease. The treatment of three categories of patients will be discussed viz. Treatment of early breast cancer, treatment of local advanced disease and treatment of advanced disease.

Treatment of early (potentially curable) breast cancer

- Early breast cancer is defined as those cases categorized as stages I and II in UIC staging.
- Local eradication of the disease is the mainstay of treatment. This is usually achieved by surgery in combination of some form of adjuvant therapy as outlined below.

There is now a definite shift towards more conservative surgical techniques as there has been no actual survival benefit from the more radical procedure as evidenced from clinical trials.

Either of the following two strategies may be adopted with full explanation to the patient:

1. **Conservative breast surgery** is gaining popularity in recent years. If done for properly indicated patients, the operation provides good results that are equal to radical mastectomy, while the breast is preserved minimizing psychological trauma.

The term therapy is used because it includes a combination of surgery and radiotherapy.

Wide local excision with a 2 cm safety margin. If the lesion is close to the skin, part of it may be excised to ensure the required safety margin.

Sentinel lymph node biopsy or level I sampling of axillary lymph nodes. If the nodes prove to be positive (harbouring metastases) then axillary lymph node dissection is done. If axillary lymph nodes are negative there is no need to axillary dissection.

Postoperative radiotherapy for 4 weeks directed to the whole breast with a boost at the site of the operation in order to kill any residual malignant cells.

The operation is suitable for:

- Small tumour ≤ 4 cm.
- Sometimes large lesions (up to 5 cm) in large breasts.
- Peripheral lesions.

Contraindications

- Pregnancy
- In situ breast cancer more than 20% due to the common incidence of multicentricity.
- Large or central tumours in small breasts (no cosmetic advantage).
- Multicentric disease as detected by soft tissue mammography.
- Collagen vascular disease (poor tolerance to radiotherapy).

Further management

Hormonal therapy (HT) for all hormone receptor positive cases. It reduces ipsilateral and contralateral breast recurrence by 40%. Tamoxifen blocks estrogen receptors and anastrozole is an aromatase inhibitor which inhibits peripheral conversion of androgen to estrogens.

Chemotherapy is indicated for

1. Positive axillary nodes.
2. All patients below 70 years.
3. Tumours more than 1 cm.
4. Hormone receptor negative and Her2/neu positive tumours (denote aggressive tumours).

Targeted therapy for Her2/neu positive cases. Monoclonal antibodies are given against Her2/neu receptors (herceptin)

2. **Modified radical mastectomy, MRM** (of Patey). The operation is indicated for large, central, multicentric tumours or local recurrence after conservative surgery. A skin ellipse over the tumour with at least 5 cm safety margin, including the areola and nipple is fashioned. The whole breast including the tumour is excised. To remove the whole mammary tissue, skin flaps are raised to the midline medially, the clavicle superiorly, the anterior border of the latissimus dorsi laterally, and the upper 1/4 of the anterior rectus sheath inferiorly. The axilla is cleared, i.e. all lymph nodes medial to the axillary vein and the axillary fat are removed in continuity with the excised breast. The axillary vessels, the nerve to serratus anterior, and the nerve to latissimus dorsi are spared.

Postoperative radiotherapy to the internal mammary and supraclavicular lymph nodes is advised for patients with positive axillary lymph nodes and for patients with tumours in the medial half of the breast. Postoperative radiotherapy does not improve survival, but it does reduce the incidence of local recurrence.

Treatment of intermediate disease (locally advanced breast cancer)

This category of patients include tumours larger than 5 cm or fixed axillary or internal mammary lymph nodes. Distant metastases should be excluded by a methodical workup including CT scans, bone scans or PET-CT scan.

Treatment

Neoadjuvant chemotherapy (a regimen of preoperative chemotherapy) is given with the aim of down-staging of the tumour. If the tumour can be down staged, a form of breast conservative surgery is done. If a poor response is noticed, modified radical mastectomy may be done.

Further management proceeds as under conservative breast surgery.

Treatment of advanced disease

This category comprises stages III and VI UICC, T4 patients, fungation, ulceration, inflammatory carcinoma or recurrent cases as well as distant metastases are included in this category. The aim is palliation and improving the quality of life.

Modalities of treatment

1. Radiotherapy

- a. It is used locoregionally for palliation of pain or ulceration.
- b. Systemically for brain metastases to decrease edema and intracranial tension, for spinal metastases to decrease local edema and spinal cord compression and for bone secondaries and pathological fractures.
- c. Superior vena cava obstruction.

2. Hormone therapy The following issues are relevant

- a. It is only given to hormone receptor positive patients, with a 60-70% response.
- b. It is more effective in postmenopausal women.
- c. It is not very effective in hormone negative patients (response, 10%), with visceral metastases or young patients below 35 years.
- d. Tamoxifen, the primary antioestrogen is given for not more than five years to avoid the risk of endometrial cancer or thrombogenicity. If no response to tamoxifen, aromatase inhibitors may be used.

3. Chemotherapy The following issues are relevant

- a. It is the basic treatment of metastatic disease; response 60-80%.
- b. It can be used as a targeted therapy for Her2/neu positive cases.
- c. It is given for visceral metastases as the liver, lungs or brain, for hormone negative patients and failure of hormone therapy.

4. Surgery is indicated for the following cases

- a. Simple mastectomy to remove an unpleasant, malodorous fungating tumour.
- b. Internal fixation of pathological fractures.
- c. Urgent decompression and stabilization of the spine if a vertebral collapse causes spinal cord compression.

Follow-up

After treatment, patients are reviewed at regular intervals, usually 3-monthly for the first 2 years, 4-monthly for the next 3 years, and annually thereafter. This is required to:

1. Detect and treat complications of mastectomy:

- a. Psychiatric morbidity caused by the loss of the breast which is a symbol of femininity, is common.
 - b. Arm edema results from excision of lymphatics, their obstruction by radiotherapy, lymphangitis caused by infection, or malignant axillary recurrence blocking them. Very early post-operative edema may also be caused by thrombosis of the axillary vein. Avoidance of radiotherapy to the axilla which has been surgically evacuated of its nodes reduces the possibility of lymphatic edema. The patient is warned to avoid minor trauma to the ipsilateral hand and should wear gloves when carrying out rough work in order to avoid infection and lymphangitis. When arm edema develops, it is difficult to treat. Arm elevation, massage, and elastic or pneumatic arm compression are partially effective.
2. **Detect local recurrence or distant disease.** Because of the incidence of cancer in the other breast (1% per year) annual mammography of the contralateral breast is done.
 3. **Instructions.** Patients are instructed not to get pregnant for at least three years, and to use non-hormonal contraception, to avoid the stimulating effect of hormones on possible residual tumour.

Reconstructive procedures after mastectomy

There is now great tendency to offer the patient who had mastectomy, some form of reconstructive surgery so that there will be minimal cosmetic deformity. Two procedures are available.

1. **A synthetic implant** may be inserted behind the pectoralis major muscle and it is inflated gradually until it attains the desired size.
2. **A myocutaneous flap** is used to reconstruct the breast. The transverse rectus abdominis myocutaneous flap (TRAM) is very efficient. The idea is take a transverse flap of the skin of the lower abdominal wall together with the rectus muscle with the blood supply based on the superior epigastric vessels. The flap is transposed upwards to form a breast substitute.

Management of specific problems

- **Hypercalcaemia** is an oncological emergency that occurs in patients with widespread bone metastases. Patients complain of thirst, drowsiness, and constipation. The patient is dehydrated. Serum calcium is high. Treatment is correction of dehydration by IV fluids, together with frusemide, prednisolone and biphosphonates.
- **Pathological fractures.** The limb should be immobilized and internal fixation is used whenever clinically indicated. The patient is given radiotherapy to the fracture site.
- **Cerebral metastases** are treated by a combination of corticosteroids and radiotherapy. Occasionally a solitary brain metastasis is suitable for surgical excision. The prerequisites are well controlled breast primary, a long life expectancy, and an accessible area in the brain.
- **Spinal cord compression** requires urgent surgical cord decompression, with stabilization, followed by radiotherapy.

- **Superior vena cava** obstruction is life threatening and requires urgent treatment. The classical manifestations are dyspnoea, possibly with cyanosis, and dilated veins in the head and neck. Radiotherapy is the treatment of choice.
- **Pleural effusion commonly** responds to systemic therapy and chest tube drainage. If not, local instillation of the cytotoxic bleomycin through the tube may be required.
- **Liver metastases** are generally regarded as an indication for chemotherapy.

Prognosis

- Prognosis of patients with carcinoma of the breast depends on the following factors
1. **Type of the tumour.** The best prognosis is provided by the in situ carcinoma, and Paget's disease, while the worst is the inflammatory carcinoma. The common invasive duct carcinoma is of intermediate prognosis.
 2. The **T stage** of the primary tumour. The higher the T stage, the worse is the prognosis.
 3. **Number, size, mobility, and location** of the involved lymph nodes. Assessment of lymph node involvement depends on the histological examination as the clinical evaluation is very inaccurate in this regard.
 - Large fixed nodes are of bad prognosis.
 - The number of involved nodes largely affects the prognosis.
 - Patients with negative axillary nodes have a 10-year survival rate of 65%.
 - Patients with 1-3 positive axillary nodes have a 10-year survival rate of 38%.
 - Patients with more than 4 positive axillary nodes have a 10-year survival rate of 13%.
 - The prognosis worsens the higher the affected nodes in the axilla. Involvement of level III nodes carries a bad prognosis.
 4. The presence of **distant metastases** markedly worsens the prognosis.
 5. **Hormone receptor status.** Receptor positive tumours respond more often to hormonal therapy and have a better prognosis than those that are receptor negative.
 6. The **site of the tumour.** Medial half tumours have a worse prognosis than those of the lateral half due to early involvement of the internal mammary lymph nodes.
 7. Measurement of tumour proliferation by **thymidine labelling** index and measurement of oncogene products.

Diseases of the male breast

Gynaecomastia

Gynaecomastia is generalized enlargement of the male breast. It may be unilateral or bilateral (Fig. 27.20).

Aetiology

Most cases are related to an imbalance between oestrogens and androgens.

Physiological gynaecomastia

- **Infantile gynaecomastia.** Circulating maternal sex hormones cause gynaecomastia in a small number of male neonates. This usually resolves within six months.

- Pubertal gynaecomastia. It occurs in up to 70% of normal pubertal boys and is often asymptomatic. It usually resolves within 2 years when adult testosterone levels are reached.
- Senile gynaecomastia. The increased prevalence of gynaecomastia with age is related to the reduction of testicular function.

Secondary gynaecomastia

- Reduced production of testosterone as after orchidectomy for prostatic cancer, and testicular atrophy after viral orchitis and leprosy.
- High levels of oestrogen caused by feminising testicular tumour and suprarenal tumours.
- Failure to metabolize oestrogens in cases of chronic liver disease
- Drugs as oestrogen preparations, and long term administration of cimetidine, digoxin, spironolactone, phenothiazines, and cannabis.
- Ectopic hormonal production in bronchogenic carcinoma.



Fig. (27.20) Gynaecomastia may be unilateral or bilateral

Clinical features

- The usual complaint is breast enlargement which may be unilateral or bilateral, and with or without tenderness.
- Examination reveals a subareolar mass (a disc of tissue) which is soft and mobile. Any eccentricity, induration, or fixation should raise the suspicion of malignancy (this is not a possibility in children and adolescents). Examination should include the abdomen and testes.

Investigations

These are not required for neonatal and adolescent cases.

1. Liver function tests, and hormone assays in appropriate cases.
2. Biopsy in cases suspected of malignancy.

Treatment

- **Most cases** require no treatment. Reassurance is all that is required for physiological gynaecomastia. The neonatal and adolescent types usually resolve spontaneously.
- Secondary gynaecomastia will often improve following treatment of the underlying condition or withdrawal of the responsible medication.
- Persistent gynaecomastia causing embarrassment is treated surgically by subcutaneous mastectomy.



Fig. (27.21) Male breast cancer

Carcinoma of the male breast

- Male breast cancer is rare.
- Because of the deficiency of breast tissue it rapidly becomes attached to the skin and chest wall, and ulceration is common (Fig. 27.21).
- The main differential diagnosis is from gynaecomastia.

- Staging and treatment are similar to that for carcinoma of the female breast, but castration is the principal means of hormonal control of advanced cases.
- Prognosis is worse than that of female breast cancer.

Fig. (27.22) is an algorithm for the management of a clinically doubtful breast mass

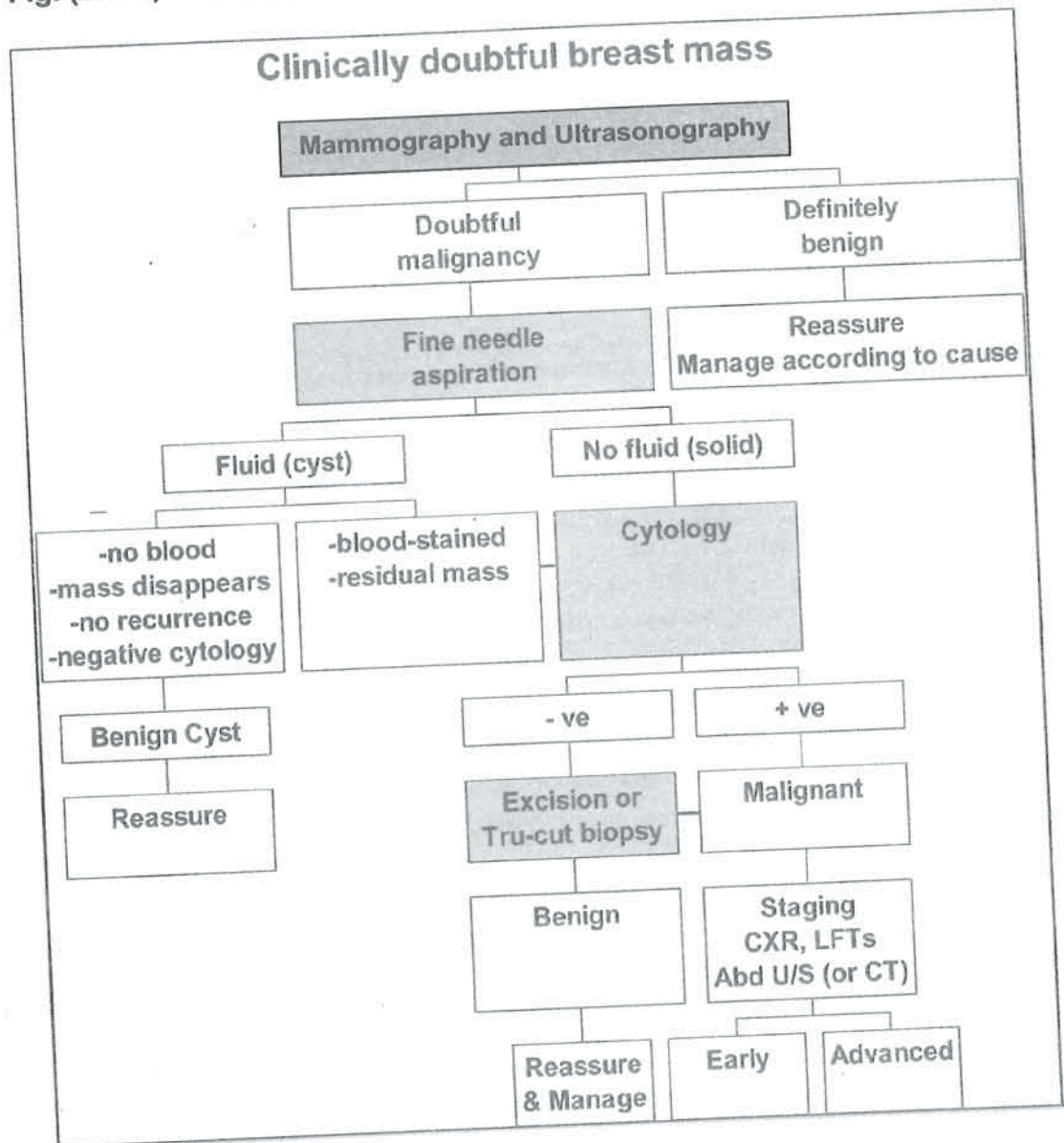


Fig. (27.22) Algorithm for the management of a breast mass

CARDIOTHORACIC SURGERY

Chest injuries

In civil practice, chest injuries are relatively uncommon, and are mainly due to car accidents and stab wounds. In war, they are very common, accounting for nearly 10% of all casualties and for about 25% of all fatalities, and are usually caused by missiles which often involve other parts of the body.

Aetiology and mechanism of injury

1. **Blunt trauma.** Such trauma is caused by blows to the chest wall, fall from a height, or motor car accidents in which crushing of the chest wall occurs.
2. **Penetrating trauma.** The penetration is caused by stabs, or gunshots. The kinetic energy of the bullet is proportional to its mass multiplied by its square velocity.
3. **Blasts.** The rapidly expanding sphere of high-temperature, high-pressure gaseous products constitutes the blast wave. This causes stress waves which disrupt the alveolo-capillary membrane resulting in haemorrhage and leakage of interstitial fluids into the alveoli. Clinically the patient will complain of dyspnoea and cough with frothy bloody sputum. In addition to the blast wave, the produced missiles from the explosion may cause blunt or penetrating injuries to the chest.

Possible consequences of chest trauma

1. Fractured ribs simple fractures and flail chest.
2. Haemothorax.
3. Pneumothorax.
4. Contusion or laceration of the lung.
5. Cardiac injuries.
6. Injuries to the major blood vessels.
7. Diaphragmatic injuries (Fig. 28.1).
8. Oesophageal injuries (Chapter 29).

Ventilation may be seriously affected in chest injuries due to the following factors

1. Severe pain due to rib fractures.
2. Instability of the chest wall due to the presence of a flail segment.
3. Lung collapse due to haemothorax or pneumothorax.
4. Pulmonary contusion.
5. Accumulation of secretions in the tracheobronchial tree.
6. Depression of the respiratory centre due to associated head injury.

CHAPTER CONTENTS

- Chest injuries
- Empyema
- Bronchogenic carcinoma
- Pulmonary metastases
- Postoperative pulmonary complications
- Mediastinal tumours
- Card/ac operations
- Cardiac arrest
- Principles of surgery for congenital heart diseases
- Principles of surgery for acquired valvular heart diseases
- Ischaemic heart disease
- Thoracic aortic aneurysms
- Aortic dissection



Fig. 28.1. Mechanism of diaphragmatic injury.

REMEMBER

- A stab wound or a bullet injury to the lower chest may cause an associated abdominal injury.
- In chest injuries, the most common cause of death between the site of accident and the emergency room is respiratory insufficiency which presents mainly by restlessness and agitation.
- The surest method to diagnose hypoxia is to do arterial blood gases.

First aid measures

See chapter 2 for details of primary and secondary surveys for trauma patients.

- Check that the upper airway is patent.
- A tension pneumothorax should be deflated by inserting a needle in the second intercostal space. Otherwise, venous return stops.
- A sucking chest wound is immediately sealed by a dressing, which is fixed at 3 sides.
- If there is severe pain, a strong analgesic should be prescribed.
- A flail segment should be initially stabilized by external strapping (Fig. 28.2) and later by endotracheal intubation and positive pressure ventilation.
- Removal of tracheobronchial secretions by encouraging the patient to cough or by bronchoscopic aspiration.
- Emergency thoracotomy may be required if there is uncontrollable internal or external bleeding.
- In serious chest injuries it is necessary to admit the patient to the ICU where an endotracheal tube is inserted. The endotracheal tube will allow effective and repeated aspiration of the tracheobronchial tree. If the patient is hypoxic or has flail chest, the endotracheal tube will be combined with intermittent positive pressure respiration.
- After the first aid measures have been performed and the patient's condition is stabilized (primary survey), a more meticulous examination is done and the necessary investigations are performed (secondary survey). Individual injuries are then dealt with.
- Look for associated injuries.



Fig. 28.2. Strapping is the first aid measure for a flail chest.

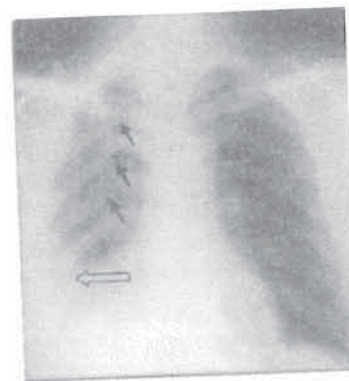


Fig. 28.3. Multiple rib fractures. Notice that they all occur posteriorly at the rib angles. Right haemothorax shows as an opacity that obliterates the costophrenic angle and rises in the axilla.

Rib fractures**Aetiology**

1. Direct violence, e.g. a blow or crush, may produce a fracture at any site. Visceral injury is common as the broken ends are driven inwards. In severe crushes, severe ribs may sustain double fractures with depression of the fragments and paradoxical movement (flail chest).
2. Indirect violence by compression of the chest bends the ribs beyond their natural elasticity so that they give way at the sites of maximum stress (burst fractures). The rib usually breaks at the angle (Fig. 28.3), and since the broken ends are driven outwards visceral injury is uncommon.
3. Muscular violence, e.g. during violent sneezing, coughing or lifting a heavy weight may cause fracture of the anterior segment of a rib in old people (senile osteoporosis).

Simple rib fractures

- These are usually due to direct violence.
- Clinically there is pain, tenderness and possibly crepitus at the fracture site.
- The severe pain associated with multiple fractures may limit respiratory expansion especially in elderly persons, thus leading to atelectasis and infection.
- Simple rib fracture can be lethal if there is undetected underlying haemothorax or pneumothorax.

- Plain X-ray of the chest is essential to detect any air or fluid collection in the pleural space.
- **Treatment.** Relief of pain is essential to allow the patient to expand the lungs properly. Strong **analgesics** as pethidine or nonsteroidal anti-inflammatory drugs (NSAID5) are prescribed as necessary. Intercostal nerve block may be required for persistent pain. In the elderly, an elastic corset may be useful.

Flail chest

- This occurs when more than four ribs are fractured at two points due to a crushing injury of the chest (Fig. 28.4).
- The integrity of the chest wall is lost and the flail segment will move in a paradoxical way to the rest of the chest wall. During inspiration, the flail segment will be retracted, and vice versa, (Fig. 28.5). This **paradoxical movement** limits the patients ability to create a negative intrathoracic pressure to ventilate the lungs and so it markedly interferes with the respiratory efficiency and leads to sputum and CO₂ retention.
- An additional factor is the side to side movement of the heart and mediastinum (Mediastinal flutter) which interferes with the venous return.
- Patients with flail chest usually have underlying **pulmonary contusion**.
- **Treatment**
 - If the flail segment is small, external immobilization of this segment by a cotton pad and adhesive plaster is enough.
 - In patients with severe paradoxical respiration especially if they are elderly, the best policy is to introduce an endotracheal tube and start intermittent positive pressure breathing (IPPB) which will lead to internal stabilization of the flail segment. If the period of required ventilation is preferable to do a tracheostomy to avoid laryngeal stenosis due to prolonged endotracheal intubation.
 - If there is an indication for thoracotomy, the fractured ribs may be secured by stainless steel wires or by special nails. This will accelerate the recovery of the patient.

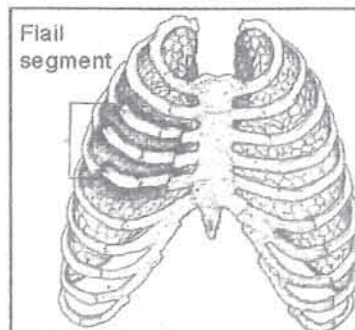


Fig. 28.4. Flail chest.

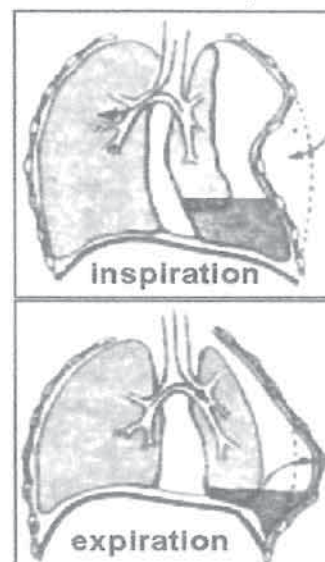


Fig. 28.5. Paradoxical movements of a flail chest

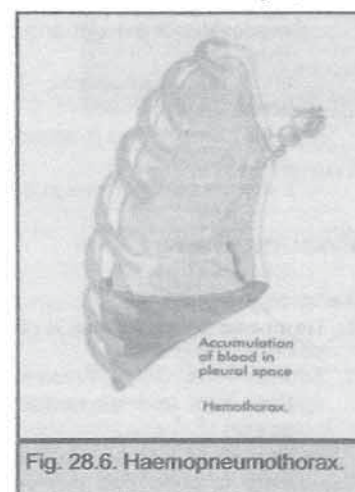


Fig. 28.6. Haemopneumothorax.

Haemothorax

Aetiology

1. Traumatic due to closed or penetrating injuries. The usual source of bleeding is injury to the intercostal or internal mammary vessels. Bleeding from a lung laceration is not profuse because the pressure in the pulmonary circulation is low, and when the lung is

- compressed by the haemothorax, the bleeding vessels are compressed.
2. **Post-operative** following cardiac, pulmonary and oesophageal operations. Insertions of a central venous line may be followed by haemothorax.
 3. **Pathological**
 - a. Tumours of the lungs, pleura or mediastinum.
 - b. Leaking aortic aneurysms.

Pathological sequelae

1. The respiratory and cardiac movements defibrinate the blood, so that the collection remains fluid in the majority of cases. Still, blood may clot making its aspiration difficult.
2. Blood in the pleural space is an irritant. It excites the formation of a considerable effusion that is rich in proteins.
3. In neglected cases a fibrin layer is deposited on both layers of the pleura. Later this deposit is transformed to a fibrous layer. The end result will be a collapsed lung and deformity of the chest wall with crowding of the ribs.
4. Blood is an excellent culture medium and infection is relatively common.
5. In traumatic cases there is commonly an associated pneumothorax i.e., haemopneumothorax (Fig. 28.6).

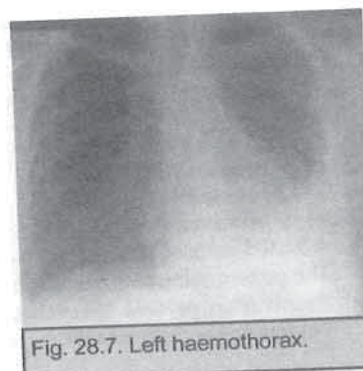


Fig. 28.7. Left haemothorax.



Fig. 28.8. Left haemopneumothorax.

Clinical features

- Chest pain and dyspnoea.
- Clinical picture of hypovolaemia.
- Chest examination reveals evidence of fluid in the pleural space, i.e., diminished lung expansion and dullness. In case of massive haemothorax the mediastinum may be shifted to the opposite side.

Investigations

- **Plain chest x-ray** If the haemothorax is less than 500 ml, it will lead only to obliteration of the costophrenic angle. Larger amounts will lead to an opacity rising to the axilla (Fig. 28.3 & 28.7). Haemopneumothorax (a combination of blood and air accumulation in the pleura) shows as a transverse air-fluid level (Fig. 28.8) and lung collapse.
- **Intercostal aspiration** reveals blood.

Treatment

The aim is to drain the blood as early as possible to allow full lung expansion and so avoid the harmful effects of haemothorax.

- Insert a cannula and correct the hypovolaemia.
- If there is pain, an analgesic is prescribed.
- Definitive treatment is by inserting a chest tube. The tube is usually inserted in the fifth intercostal space in the mid-axillary line. It should then be connected to an underwater seal. The chest tube will allow drainage of blood and at the same time it allows follow-up of the patient. So long as the patient is stable, the amount of drained blood decreasing and the lung is expanding, this means that conservative treatment

successful. The chest tube should be kept until no more drainage occurs and the lung is fully expanded, when the tube should be removed.

▪ **Indications for thoracotomy**

- Drainage of more than 2000 ml of blood after insertion of the tube.
- Bleeding more than 200 ml/hour.
- Clotted haemothorax. This is diagnosed by failure of the tube to drain the blood. These patients need thoracotomy for evacuation of the clot and decortication of both the lung and chest wall.
- Loculated haemothorax. The tube partially evacuates the blood.
- If the haemothorax is associated with other injuries, e.g., of the oesophagus or cardiac tamponade.
- In the presence of foreign bodies.

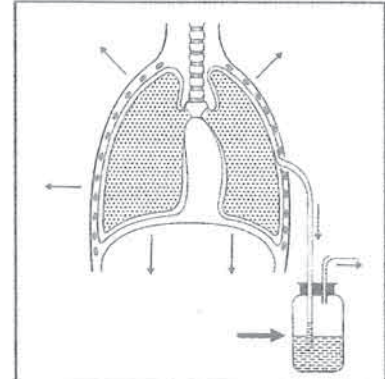


Fig. 28.9. Intercostal chest tube for drainage of fluid or air from the pleural cavity. The tube is attached to an under-water seal (arrow).

Pneumothorax

Aetiology (Fig. 28.10)

1. Traumatic pneumothorax is caused by either blunt or penetrating injuries of the chest.
2. Spontaneous pneumothorax is due to
 - a. Rupture of an emphysematous bulla, solitary lung cyst or cystic lung.
 - b. Rupture of a small subpleural tuberculous cavity. In this case there is underlying pulmonary tuberculosis and hydropneumothorax usually results.
3. Iatrogenic pneumothorax is due to
 - a. Positive pressure ventilation that is complicated by rupture of alveoli (barotrauma).
 - b. During insertion of a central venous line in the neck.

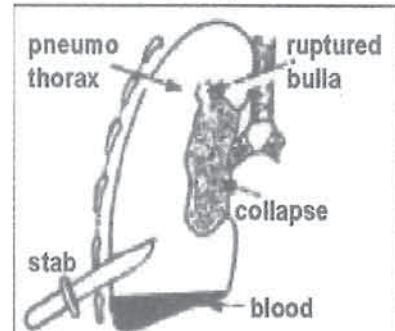


Fig. 28.10. The commonest causes of pneumothorax are ruptured emphysematous bulla and injuries. In the latter accumulation of blood is a common association.

Types

1. Simple pneumothorax.
2. Open pneumothorax.
3. Tension pneumothorax.

Simple pneumothorax

- A limited amount of air is introduced in the pleural cavity.
- The patient complains of chest pain and slight dyspnoea.
- Examination reveals diminished air entry, hyperresonance but there is no mediastinal shift.
- Plain chest X-ray reveals translucency and absence of lung markings, the edge of the collapsed lung is usually visible.
- Treatment
 - If the amount of air is small and there is no dyspnoea, the patient is treated conservatively until spontaneous absorption of the pneumothorax takes place.
 - If the patient is dyspnoeic, an intercostal chest tube is inserted until expansion of the lung becomes complete.

Open pneumothorax (sucking chest wound, Fig. 28.11)

- This occurs secondary to a wound in the chest wall leading to a communication between the pleural space and the atmosphere.
- When the patient inspires, some air will be inspired through the trachea but a percentage will pass through the pleuro-cutaneous wound. If the chest wound is large, all the air will pass through the pleurocutaneous wound resulting in collapse of the lung and practically no intrapulmonary ventilation.
- The mediastinum shifts back and forth with each breath and the cardiac output is reduced (mediastinal flutter).
- Clinically open pneumothorax is diagnosed by the sound of air going through the defect.
- **Treatment**
 - Open pneumothorax is an emergency and should be treated immediately by applying an adhesive external dressing which
 - The wound is then repaired in theatre, and an intercostal chest tube is inserted to drain the plural air.

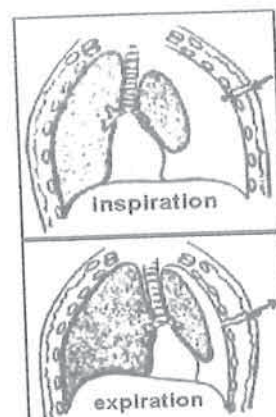


Fig. 28.11. Open pneumothorax. Notice the side to side movements of the mediastinum.

Tension pneumothorax (Fig. 28.12 & 28.13)

- A small puncture wound of the chest wall involving the visceral pleura, may produce a valve action in the visceral pleura and allows air to enter the pleural cavity during inspiration but prevents its exit during expiration.
- The lung gradually collapses and the mediastinum shifts to the opposite side.
- The patient becomes severely dyspnoeic due to compression of the opposite lung, and respiratory arrest may follow.

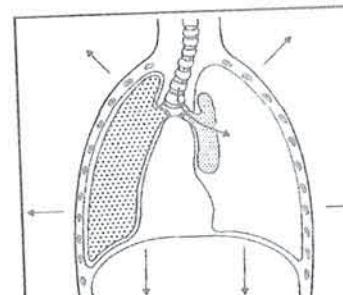


Fig. 28.12. Tension pneumothorax.

Tension pneumothorax is rapidly fatal.

- Examination reveals diminished air entry, hyper-resonance, shift of the trachea and mediastinum and engorged neck veins.

N.B. Tension pneumothorax is a cause of obstructive shock because it prevents cardiac filling.

Treatment

- Tension pneumothorax is an emergency. There is no time for X-ray diagnosis. A wide-bore needle should be immediately inserted in the second intercostal space to allow decompression of the pneumothorax.
- Later, an intercostal chest tube connected to an underwater seal is inserted.
- Continuous bubbling of air through the intercostal tube denotes the possibility of a bronchopleural fistula.



Fig. 28.13. Plain X-ray shows left tension pneumothorax. Seeing the patient as such to radiology room is wrong. It may cost life. The case should be diagnosed clinically and immediately deflated by a wide bore needle. Notice marked mediastinal shift and rib fractures.

Pulmonary contusion

- Pulmonary contusions occur as a result of impact injuries to the chest wall.
- They lead to outpouring of fluid in the walls of the alveoli and into the alveolar spaces with the development of pulmonary oedema and pneumonitis.
- Disturbance occurs in the ventilation perfusion ratio in the contused areas leading to diminished oxygenation of the blood and eventually tissue hypoxia occurs.
- Treatment is conservative by antibiotics and physiotherapy.

Cardiac injuries

Injuries to the heart are usually due to penetrating wounds, e.g., stabs and gun-shot wounds, and rarely due to closed injuries, e.g. crushing of the chest and fractured ribs.

Clinical features

Cardiac injury may manifest itself by

1. Profuse external haemorrhage which may come from the pericardial vessels, cardiac muscle, coronary vessels, heart chambers or great vessels.
2. Massive haemothorax.
3. Cardiac tamponade due to bleeding into the pericardial sac which compresses the heart prevents diastolic filling and reduces cardiac output. As a result
 1. Neck veins are engorged.
 2. Heart sounds are faint.
 3. Blood pressure is reduced.
 4. Pulse is weak.
 5. Cardiac dullness is enlarged.

The first three signs are called Beck's triad.

4. Injury of thoracic aorta may be rapidly fatal if it opens in the pleura. However, if it is contained in the mediastinum there is enough time to reach hospital and do plain radiography. The condition is suspected by finding a wide mediastinum on a PA view of a chest X-ray (Fig. 28.14).

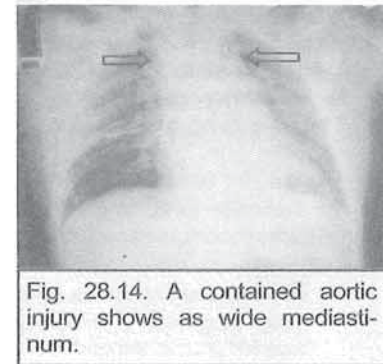
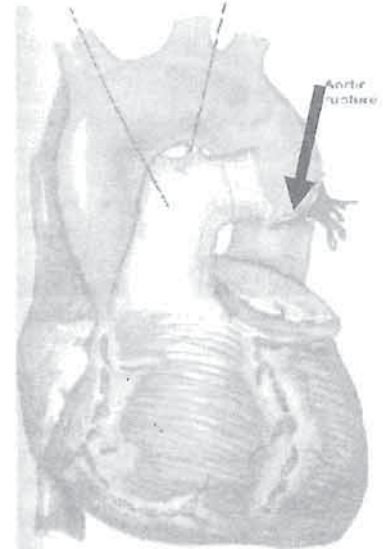


Fig. 28.14. A contained aortic injury shows as wide mediastinum.

Treatment

1. Initial life-saving treatment. In cardiac tamponade, immediate aspiration of the pericardium is necessary to obtain temporary relief until operation is carried out (Fig. 2.6 & 28.15). The aspiration cannula is left in place to keep the heart decompressed till definitive measures are taken.
2. Definitive treatment. In all cases, the heart is exposed through a left thoracotomy as soon as possible. Blood is evacuated, bleeding vessels are ligated, and tears in the myocardium are repaired. The pericardium should be left open to allow any effusion to escape into the pleura which should be drained.

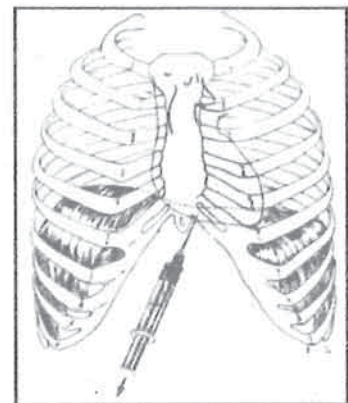


Fig. 28.15. Aspiration of pericardial blood that causes tamponade

Diaphragmatic Injuries

- More common in penetrating chest wounds.
- More common on the left side, trauma to the left chest wall should raise suspicion of the injury.
- Usually associated with herniation of abdominal viscera into the chest.
- Abdominal tenderness, dyspnea, shoulder pain diminished breath sound and audible intestinal sounds on the chest.
- Plain x-rays of the chest and chest CT are diagnostic.
- Abdominal repair.

Beck's triad is seen in cases with cardiac tamponade.

1. Neck veins are engorged.
2. Heart sounds are faint.
3. Blood pressure is reduced.

Esophageal injuries (perforations) may have a potentially serious outcome

Causes

1. Iatrogenic, during endoscopic dilatation of strictures (the commonest cause).
2. Penetrating or blunt injuries to the neck or chest.
3. Swallowing of corrosives or foreign bodies.
4. Spontaneous perforation, due to forceful vomiting (Boerhaave's syndrome) (perforations of tumours are rare).

Clinical features

- Acute onset of pain at the site of rupture.
- Acute onset of fever, tachycardia and hypotension.
- Mediastinal emphysema which presents as subcutaneous emphysema of the neck or chest wall. Mediastinitis is rapidly fatal if not urgently treated.
- Pneumothorax and/or pleural effusion.

Investigations should be urgent

- Plain X-rays mediastinal emphysema or hydropneumothorax.
- Water soluble swallow may reveal the perforation.

Management

- Management of shock, i.v. antibiotics and nil by mouth are essential. Do not pass a nasogastric tube.
- Early cervical perforations surgical closure and external drainage, while late perforations are managed by external drainage and i.v. hyperalimentation.
- Early thoracic perforations surgical repair and chest drainage, while late perforations are managed by esophageal resection.
- Perforations of the abdominal esophagus must be treated surgically.

Table (28.1) illustrates the radiological finding in thoracic injuries

Table (28.1): Radiological finding in thoracic injuries

Lungs, pleura	Pneumothorax, haemothorax, pulmonary contusion
Mediastinum	Widening of the mediastinum suggests aortic rupture. Air in the mediastinum suggest esophageal perforation.
Diaphragm	Diaphragmatic rupture on the left side with herniation of abdominal viscera
Bones	Rib fractures, flail segments. Fractures of the first rib or sternum denote high velocity injuries with serious outcome. Fractures of the lower ribs raise the probability of splenic or liver injury

Empyema

An empyema (pyothorax) is a collection of pus in the pleural cavity. It may be acute, chronic non-specific or tuberculous.

Acute empyema

Aetiology

Routes of infection

Acute empyema is commonest in children as a complication of lobar pneumonia or bronchopneumonia, but may arise at any age from infection of the pleura by any of the following routes

1. Direct access through an open wound, as in posttraumatic and post-operative empyema.
2. Local spread from the chest wall (osteomyelitis of a rib), lung (pneumonia, lung abscess, bronchiectasis or carcinoma), oesophagus (perforation or carcinoma) or diaphragm (subphrenic or liver abscess).
3. Blood spread rarely occurs as a complication of septicaemia and pyaemia.

Causative organisms are pneumococci (commonest), streptococci and staphylococci. Occasionally Gram negative bacilli and anaerobes are responsible.

Clinical features

There is usually a history of chest infection followed by pain in the chest with dyspnoea, cyanosis, fever and other signs of severe toxæmia. The diagnosis is established by:

1. Examination of the chest which reveals signs of pleural effusion, viz., stony dullness, absence of breath sounds and tactile fremitus and displacement of the mediastinum to the opposite side.
2. X-ray examination shows a uniform opacity tapering towards the axilla and displacing the heart to the opposite side.
3. Aspiration confirms the diagnosis and allows identification of the organism by culture and antibiotic sensitivity tests.

Treatment

The definitive treatment of an abscess anywhere in the body is by drainage of pus. This is achieved in three ways

1. Repeated aspiration is employed in early stages and is supported by antibiotics according to culture and sensitivity.
2. Closed drainage through an intercostal chest tube is carried out if the fluid collects rapidly or if the pus becomes too thick to be aspirated. A self-retaining catheter is inserted through a trocar and cannula and connected to an underwater seal.
3. Open drainage by rib resection is indicated only when full localization has occurred.

Chronic non-specific empyema

Definition

Chronic empyema is defined as pleural sepsis with failure of the lung to expand after evacuation of the pus.

Types

- **Open chronic empyema (chronic empyema sinus)** is due to mismanagement of acute empyema. The chief causes are:

- Faulty drainage, which may be too early (collapse of lung), too late (rigid pleura), too high (independent drainage) or too low (opening blocked by the diaphragm).
 - Inadequate post-operative care, e.g., early removal of the tube and omission of lavage or breathing exercises.
 - Underlying disease in the chest wall (e.g., osteomyelitis of a rib); pleura (e.g., foreign bodies, or bronchopleural fistula); or lung (fibrosis, bronchiectasis, lung abscess or tumour).
 - Poor general condition, due to anaemia, toxæmia or debility.
- **Closed chronic empyema** may be:
- An encysted empyema developing insidiously as a closed collection of pus completely walled off by adhesions.
 - A recurrent empyema discharging intermittently into a bronchus through a bronchopleural fistula.

Clinical features

1. In most cases there is a chronic sinus in the chest wall leading to the pleural cavity and discharging pus, either continuously or intermittently.
2. Rarely the empyema perforates an intercostal space to form a subcutaneous abscess (empyema necessitans).
3. The chronic suppuration leads to general ill-health with anaemia and clubbing of the fingers.
4. Acute exacerbations are common with fever and chest pain.
5. In long-standing cases, the progressive fibrosis causes contraction and rigidity of the chest wall with crowding of the ribs, elevation of the diaphragm, displacement of the mediastinum to the affected side and scoliotic deformity of the spine.

Diagnosis

1. Bacteriological examination of pus and sputum is carried out to exclude tuberculosis and to identify the causative organism.
2. X-ray examination of the chest may reveal foreign bodies or underlying lung disease.
3. A lipiodol pleurogram, through the sinus opening, shows the size of the cavity and the presence and site of any bronchial communication. A few crystals of Sudan added to lipoidal will appear in the sputum if a fistula is present.

Treatment

An attempt is made to obliterate the cavity by:

1. Adequate drainage that is established at a suitable level and followed by proper lavage and physiotherapy cures 70% of cases within 3 months.
2. Decortication which is complete excision of the thickened visceral pleura that forms a tight "peel" around the lung so that the freed lung can expand and obliterate the cavity.

Tuberculous empyema

Tuberculous infection of the pleura may occur as a complication of

1. Rupture of a tuberculous cavity.
2. Pleural effusion.
3. Operations on tuberculous lungs.

Treatment

- **In the absence of secondary infection:**

- Repeated aspiration.
- Anti-tuberculous treatment.
- If these fail decortication is performed.
- **Cases with secondary infection:**
 - Prolonged drainage until the secondary infection is overcome followed by decortication.
 - Associated lung disease or bronchopleural fistula is dealt with the time of decortication.

Bronchogenic carcinoma

Bronchial cancer affects both sexes. It is, however, the commonest cancer in men. It has a very bad prognosis.

Aetiology

The incidence of lung cancer has shown a marked rise during recent years, partly because of improved methods of diagnosis and partly because of two causes

1. Excessive cigarette smoking. The causative relationship is a definite one. Both active and passive smoking are implicated, and the incidence of lung cancer is proportionate to the frequency of smoking.
2. Inhalation of irritants, such as silica and cobalt dust in mines, and petrol fumes in large cities.

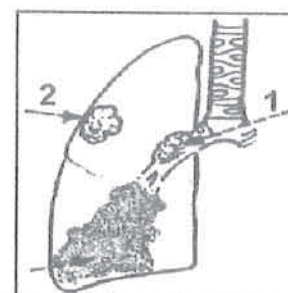


Fig. 28.16. Gross types.
1. Hilar. May cause distal lobar collapse and infection
2. Peripheral.

Pathology

Gross types (Fig. 28.16)

1. The hilar type is the commoner variety (75%). It arises in one of the main bronchi or their primary divisions, and leads to bronchial obstruction with secondary changes in the lung, such as atelectasis, pneumonia or lung abscess.
2. The peripheral type (25%) arises from the smaller bronchi and remains symptomless for a long time. This type includes the Pancoast's tumour (Fig. 28.17), which arises at the apex of the lung and invades the brachial plexus, sympathetic trunk causing Homer's syndrome, and the first rib.

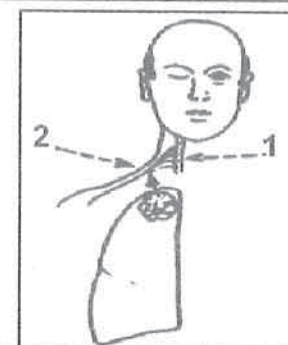


Fig. 28.17. Pancoast tumour is a subtype. It invades 1. Sympathetic chain 2. Brachial plexus. It also invades the first rib.

Histology

Three types are encountered.

1. Squamous-cell carcinoma (60%) is composed of irregularly arranged squamous cells.
2. Oat-cell carcinoma (30%) is composed of undifferentiated small cells with very scanty fibrous stroma. It is the most malignant variety, and usually occurs near the hilum.
3. Adenocarcinoma (10%) is composed of columnar cells arranged in irregular acini or papillary formations. It is commoner in females.

Spread

1. Direct to the mediastinum, pleura, chest wall or pericardium.

2. Lymphatic (Fig. 28.18) to the hilar lymph nodes and then to the tracheobronchial and lower deep cervical nodes.
3. Blood (Fig. 28.19) mainly to the liver, adrenals, brain and bones.

Clinical features

Cancer of the lung is much more common in males than females (10:1), and occurs most often between the ages of 40 and 60 years. It manifests itself by one of the following five syndromes

1. **Insidious type.** In most cases, the onset is insidious with a dry irritant cough, often ascribed to smoking, which later becomes asthmatic and associated with haemoptysis, dyspnoea, and chest pain.
2. **Acute type.** Sometimes, the onset is acute with symptoms of pneumonia which fails to resolve satisfactorily or relapses within a few days or weeks.
3. **Latent type.** In peripheral carcinoma, the disease may remain silent until discovered by mass radiography or may produce vague general symptoms, e.g. fatigue and indigestion, until distant metastases or symptoms of mediastinal invasion (mediastinal syndrome) appear. Distant deposits may produce manifestations of high intracranial tension (headache, vomiting and blurring of vision), pathological fractures, jaundice and adrenal failure.
4. **Pancoast type.** A peripheral tumour at the thoracic outlet (superior sulcus tumour) invades the brachial plexus, sympathetic trunk and upper ribs, producing a lower brachial plexus lesion with Horner's syndrome and erosion of the upper 2 or 3 ribs.
5. **Extrapulmonary non-metastatic type.** Undifferentiated tumours, particularly of the oat-cell type may elaborate hormone-like substances, toxins or antigens which produce a variety of manifestations which collectively called the "para-neoplastic syndromes". They include:
 - A Cushing-like syndrome due to ACTH production, which differs from the classical syndrome by an older age incidence, a greater frequency in males, and a more rapid clinical course.
 - Water retention due to secretion of ADH which may result in hyponatraemia, water intoxication and cerebral symptoms.
 - Hypercalcaemia caused by a parathormone-like polypeptide.
 - Tender gynaecomastia due to ectopic gonadotrophin secretion.
 - Carcinoid syndrome due to secretion of 5-hydroxy-tryptamine or hydroxytryptophane.
 - Malignant neuromyopathies which probably result from elaboration of antibodies to an antigen derived from the tumour. These antibodies may cause myasthenia, dermatomyositis or peripheral neuritis.

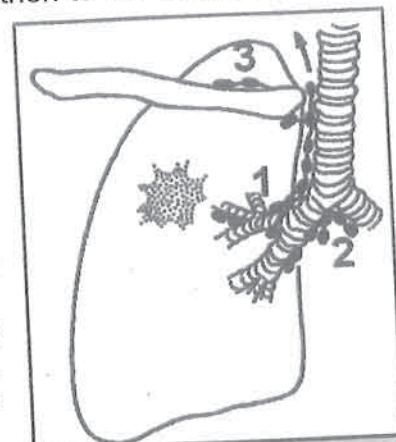


Fig. 28.18. Lymphatic spread.
1. Hilar nodes.
2. Tracheobronchial nodes.
3. Supraclavicular nodes (part of lower deep cervical group).

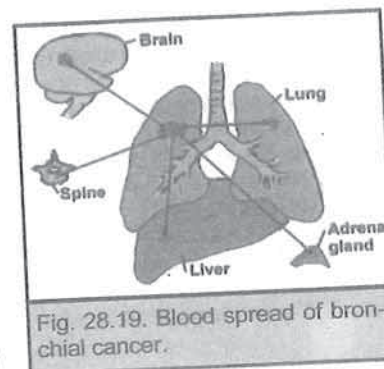


Fig. 28.19. Blood spread of bronchial cancer.

- Pulmonary osteoarthropathy may occur even with a small asymptomatic cancer. It is characterized by a sudden onset of clubbing of fingers, sometimes associated with oedema and swelling of the small joints of the hands and ankles, and pain in the knees. Removal of the cancer is followed by fast dramatic relief of joint pains and rapid subsidence of the clubbing.

Diagnosis

Keep a high index of suspicion

Bronchogenic carcinoma should be suspected in all patients over 40 who develop abnormal cough, unresolved or recurrent pneumonia, chest pain or haemoptysis. Even when the pneumonia responds to ordinary medical treatment, the chest should be X-rayed 3 weeks after cure.

Investigations

1. **Plain X-ray** may reveal a "coin" shadow (Fig. 28.20 and 28.21) or hilar opacity, or signs of obstructive emphysema, atelectasis, pneumonia, lung abscess, enlarged mediastinal glands, pleural effusion, erosion of ribs or diaphragmatic paralysis.
2. **CT scan** (Fig. 28.22) accurately localizes the tumour, and detects mediastinal lymph node enlargement. For peripheral lesions CT-guidance of needle biopsy provides a high accuracy.
3. **Bronchoscopy** (Fig. 28.23) usually establishes the diagnosis and allows assessment of operability. In about 60% of cases, the tumour is visible as a nodular necrotic mass or an irregular stenosis and a punch biopsy can be taken. Signs of inoperability include left recurrent laryngeal paralysis, compression of the trachea or widening of the carina by enlarged glands, fixity of the affected bronchus or malignant infiltration beyond the proximal centimeter of the main bronchus.
4. **Sputum cytology** may reveal the presence of malignant cells and is especially valuable when biopsy material cannot be obtained during bronchoscopy.
5. **Scalene node** biopsy may confirm the diagnosis and assess operability. The pad of fat over the scalenus anterior with the contained lymph nodes is removed and examined histologically for malignant infiltration.
6. **Needle biopsy** of a peripheral lesion under radiographic control will reveal malignant cells in about 60% of cases.
7. **Mediastinoscopy** to biopsy nodes of the paratracheal group.

Treatment

Radical resection should be carried out in operable cases. Pneumonectomy with removal of the mediastinal

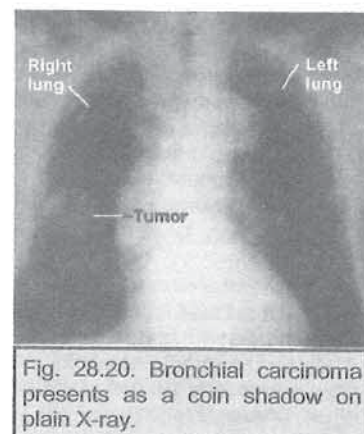


Fig. 28.20. Bronchial carcinoma presents as a coin shadow on plain X-ray.

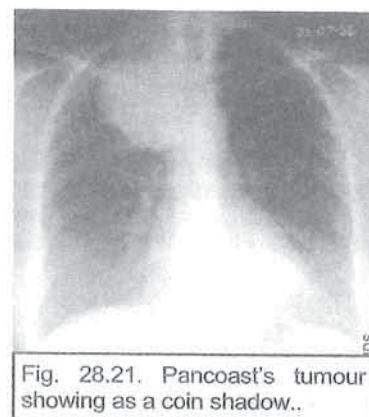


Fig. 28.21. Pancoast's tumour showing as a coin shadow..

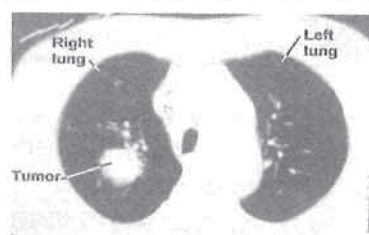


Fig. 28.22. CT scan showing bronchial carcinoma of right lung.

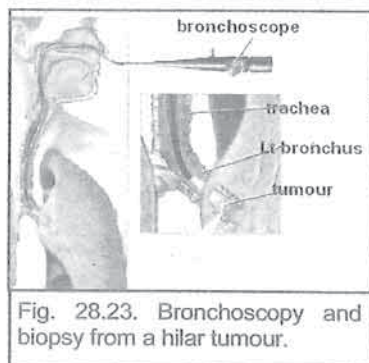


Fig. 28.23. Bronchoscopy and biopsy from a hilar tumour.

nodes gives the best results, but lobectomy may be preferred in elderly patients with peripheral carcinoma.

Palliative treatment is employed in inoperable cases.

- Palliative resection should be carried out if inoperability is discovered at thoracotomy as it relieves the chest symptoms and improves the general condition by controlling haemoptysis and suppuration.
- Radiotherapy is useful in relieving superior vena caval obstruction, bronchial obstruction and pain of skeletal metastases.
- Chemotherapy.

Pulmonary metastases

Origin

Pulmonary metastases may arise from any malignant tumour but are especially common in hypernephroma, melanoma, osteosarcoma and carcinoma of the thyroid, breast stomach and prostate.

Gross appearance

They are usually small, multiple (Fig. 28.24) and bilateral, but sometimes large and rounded (cannon-ball metastases) and occasionally central deposits near the hilum that simulate bronchogenic carcinoma.

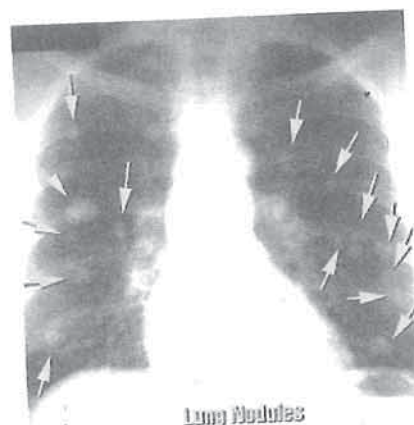


Fig. 28.24. Multiple pulmonary metastases.

Postoperative pulmonary complications

More surgical patients probably die of postoperative chest problems than anything else. Such morbidity and mortality can be largely prevented by adequate pre-and postoperative care.

Predisposing factors

Preoperative predisposing factors

1. Age of the patient. Extremes of age are more liable to complications.
2. Sex. Males are more predisposed to respiratory problems than females probably due to smoking.
3. Smoking. Heavy smokers have chronic bronchitis, and are therefore, predisposed to postoperative pulmonary complications.
4. Chronic bronchitis. This produces excessive production of mucous and damage to the cilia which lead to stagnation of mucous in the bronchial tree.
5. Dehydration. This leads to thick bronchial mucous that is difficult to expel.

Operative and post-operative predisposing factors

1. Anaesthetics. Trauma to the tracheobronchial tree, premedication with atropine and prolonged unconsciousness predispose to respiratory problems.
2. Nature of the operation. Thoracic and upper abdominal operations are more liable to complications as the wound pain interferes with deep breathing and coughing.
3. Abdominal distension, e.g. due to paralytic ileus interferes with the movement of the diaphragm and reduces the vital capacity.

4. Postoperative pain interferes with deep breathing and coughing leading to stagnation of secretions. Excess use of narcotics also interferes with deep breathing and coughing.

Types of pulmonary complications

1. Atelectasis.
2. Bronchitis.
3. Bronchopneumonia.
4. Lung abscess.
5. Pulmonary embolism (Chapter 14).
6. Mendelson's syndrome.
7. Adult respiratory distress syndrome.
8. Postoperative hypoxia.

Post-operative atelectasis (pulmonary collapse)

Predisposing factors

1. Inhalation of foreign bodies, or vomiting due to full stomach.
2. Production of tenacious mucus due to pre-operative respiratory tract infection, premedication with atropine or postoperative dehydration.
3. Inhibition of the cough reflex due to
 - a. Old age and debility.
 - b. Excessive sedation, particularly with morphine.
 - c. Operations on the chest, diaphragm or upper abdomen.
 - d. Pain.
 - e. Lack of mobility in bed.
 - f. Morbid obesity.
 - g. Recent stroke.

Pathology

Obstruction of a bronchus by a plug of mucous is followed by absorption of air distal to the obstruction and deflation of the affected area.

The consequent collapse may be

1. Lobular collapse of scattered areas throughout the lung.
2. Lobar collapse of one lobe usually the lower.
3. Massive collapse of the whole lung.

Atelectasis is the precursor of most post-operative chest complications, particularly bronchopneumonia and lung abscess.

Clinical features

Symptoms

- Atelectasis may manifest itself within 48 hours after operation, or may pass unnoticed until complicated by serious lung infection.
- In typical cases, the onset is sudden with fever in addition to dyspnoea, cyanosis and chest pain in massive cases.
- Cough is slight and the sputum is scanty and difficult to expel.
- The condition may subside after a few days with the expectoration of a considerable amount of purulent tenacious mucus.

Signs

- Unexplained tachycardia.
- A minor atelectasis produces no local signs.
- With collapse of a segment or the whole lung the affected side of the chest is flattened and immobile with dullness on percussion, diminished breath sound and displacement of the trachea and heart towards the side of collapse. As the attack subsides tubular breathing and coarse crepitations may appear.

X-ray

1. The collapsed lobe appears as a homogeneous wedge-shaped opacity.
2. Major atelectasis causes approximation of the ribs, elevation of the diaphragm and deviation of the mediastinum toward the affected side.

Prophylaxis

1. Preoperative
 - a. Eradication of respiratory infections.
 - b. Prohibition of smoking.
 - c. Breathing exercises.
 - d. Avoidance of dehydration.
 - e. Avoidance of heavy premedication, particularly with atropine and morphine.
2. Operative. Avoidance of
 - a. Trauma to the upper air passages during intubation.
 - b. Deep unconsciousness and vomiting under anaesthesia.
 - c. Tight strapping and bandaging of the chest and abdomen.
3. Post-operative
 - a. Correction of dehydration.
 - b. Avoidance of heavy sedation.
 - c. Encouragement of deep breathing, and postural exercises.
 - d. Removal of secretions by coughing, expectoration and postural drainage.
 - e. Early ambulation.
 - f. Adequate analgesia to reduce pain with coughing and deep breathing.

Treatment

1. All attempts are made to expel the obstructing plug.
 - a. Turning the patient from side to side.
 - b. Percussing the affected side of the chest.
 - c. Encouraging expectoration, if necessary by nasopharyngeal catheter.
 - d. Loosening the sputum by mucolytics, expectorants and steam inhalation.
 - e. Suction. If aeration does not occur within 6-6 hours, the bronchial plug should be removed by bronchoscopic suction.
2. Antibiotics are given to prevent infection, and breathing exercises and early ambulation are encouraged to accelerate expansion of the lung.
3. Oxygenation and if necessary intubation with mechanical ventilation of the O saturation and PO₂ are low.

Bronchitis

This is the commonest postoperative complication. Although not serious, it may predispose to more serious complications. It may occur de novo or represent an exacerbation of a pre-existing bronchitis. The patient may complain of postoperative cough with mucopurulent sputum or there may be severe suppurative bronchitis. Chest X-ray free.

Bronchopneumonia

This complication is usually a sequel to bronchitis or atelectasis. The commonest organisms are *Haemophilus influenza*, pneumococci, staphylococci or *Pseudomonas pyocyanea*. Multiple areas of patchy consolidation occur and there is profound toxemia. Chest X-ray reveals multiple areas of patchy mottling. Sputum culture will reveal the responsible organisms.

Prevention the same as prophylaxis for collapse.

Treatment

1. Once diagnosis is made the treatment is immediate physiotherapy and antibiotics.
2. Bronchoscopic removal of secretions may be required.

Mendelson's syndrome

- This syndrome is caused by aspiration of vomitus which contains irritant hydrochloric acid.
- It occurs during induction of anaesthesia in any patient with full stomach or intestinal obstruction or in pregnancy. The condition can also occur in comatose patients, e.g., after head injury or drug poisoning.
- The condition can be fatal and is clinically manifested by wheezes, cyanosis, tachycardia, tachypnoea and hypotension.
- Radiologically widespread lung infiltration occurs more on the right than on the left and more in the lower lobes. Blood gas analysis reveals severe hypoxia.
- For prophylaxis all high risk patients should have a nasogastric tube inserted before the operation for suction of the gastric contents.
- Fix treatment adequate bronchoscopic aspiration, steroids, bronchodilators and antibiotics are prescribed.

Adult respiratory distress syndrome (syn. ARDS, shock lung)

Adult respiratory distress syndrome (ARDS) is a serious condition which affects the lungs of critically ill patients. It leads to pulmonary insufficiency which is a major cause of death in affected patients.

Aetiology

1. Severe sepsis.
2. Major trauma.
3. Extensive burns.
4. Severe shock from any cause especially if requiring large volumes of intravenous fluids. Patients in septic shock are particularly at risk of developing ARDS.
5. Iatrogenic factors
 - a. Non filtered blood transfusion.
 - b. Overtransfusion of fluids.
 - c. Use of oxygen concentrations over 50%.
 - d. Massive doses of steroids.
 - e. The use of a heart lung machine for open heart surgery for a long time.
6. Lung injury due to trauma, inhalation of fumes or aspiration of gastric contents.

Pathogenesis

ARDS is supposed to be due to activation of platelets, neutrophils and macrophages leading to release of oxygen free radicals which cause:

1. Endothelial damage.
2. Edema of lung tissue.
3. Increased capillary permeability and so neutrophils and RBCs move to the inside of alveoli.
4. Impairment of O_2 diffusion.

Defects occur in the three aspects of the respiratory process:

1. **Defective ventilation**
 - a. The loss of surfactant reduces lung compliance, and, hence, its expansion.
 - b. Alveolar oedema, i.e., the presence of fluid in the alveoli prevents air from reaching the gas exchange surface.
2. **Defective perfusion**
 - a. Lung perfusion is affected if the cause of ARDS is shock.
 - b. Deoxygenated blood is shunted directly from some of the arterioles to the venular sides through opening of A-V shunts.
3. **Defective diffusion** The distance between the alveolus and the capillary, otherwise known as the alveolo-capillary membrane is thickened because of the interstitial pulmonary oedema. Oxygen and carbon dioxide find difficulty in crossing this thickened membrane.

Pathology

Macroscopic picture

- Great increase in lung weight.
- Petichial haemorrhages on epithelial surfaces.

Microscopic picture

1. Interstitial oedema and haemorrhage.
2. Alveolar oedema.
3. Perialveolar haemorrhage.
4. Inconstantly, intravascular fat globules and fibrin plugs.

Clinical progress

1. Initial state of shock, lactic acidosis and hyperventilation with low $PaCO_2$, but PaO_2 may be normal or slightly low.
2. Phase of haemodynamic equilibrium which may last several days. The patient may appear well and recovering with a normal chest X-ray but the PaO_2 is invariably low.
3. Phase of clinical respiratory distress followed by respiratory failure with confusion and occasional petechial rash. Rising $PaCO_2$ and falling PaO_2 occur despite oxygen supplement. Chest x-ray reveals bilateral pulmonary infiltrations.

Treatment

The patient should be admitted to an intensive care unit (ICU).

- Respiratory support
 - In the stage of respiratory distress, an oxygen mask is enough to improve arterial blood oxygen.
 - In the stage of respiratory failure (indicated by a $PaO_2 < 60$ mmHg) endotracheal intubation and mechanical ventilation are required.
- Treatment of the cause, e.g., correction of shock and eradication of sepsis.

Postoperative hypoxia

It is a common and serious issue. It manifests clinically with:

- Restlessness, anxiety or confusion.

- Tachypnoea.
- Tachycardia, dysrhythmias or hypotension.
- Central cyanosis is late

Common causes are interrelated

1. Pulmonary aspiration.
2. Failure to breathe deeply and cough during recovery from anesthesia.
3. Airway blockade by secretions leading to alveolar collapse.
4. Hypoventilation due to pain of upper abdominal or thoraco-abdominal incisions, opiates overdose, anesthetics or prolonged recumbency.
5. Pulmonary embolism.
6. Pulmonary edema.

Investigations

1. Pulse oximetry, a probe is attached to one finger and shows the oxygen saturation (normal values 95-100%).
2. Arterial blood gases increased PCO_2 denotes ventilation failure and decreased PO_2 denotes oxygenation failure.
3. Chest x-ray collapse, pneumothorax, consolidation.

Treatment

Treat the specific cause. The patient may need mechanical ventilation.

Mediastinal tumours**Pathology**

Mediastinal tumours are classified into two anatomical groups

1. Tumours of the anterior and superior mediastinum include retrosternal goitre, thymic tumours, dermoid cysts and teratomas.
2. Tumours of the posterior mediastinum may be
 - a. Cysts, usually of congenital origin, e.g. bronchogenic, gastrogenic and lymphatic cysts.
 - b. Enlarged lymph nodes due to secondary deposits, tuberculosis, lymphoma or leukaemia.
 - c. Solid tumours of connective tissue or neurogenic origin, e.g. neurofibroma, ganglioneuroma, and lipoma.

Clinical features

The signs and symptoms constitute the "mediastinal syndrome" due to pressure on adjacent structures. They include

1. Dyspnoea due to pressure on the air passages or encroachment on the intrathoracic space.
2. Dysphagia from compression of the oesophagus.
3. Oedema, cyanosis and dilated veins in the head, neck and upper limbs form obstruction of the superior vena cava.
4. Hoarseness of voice and brassy cough due to involvement of the recurrent laryngeal nerve.
5. Hiccup or diaphragmatic paralysis from involvement of the phrenic nerve.
6. Arrhythmias and ECG changes occur if there is invasion of the pericardium.

Diagnosis

1. Plain X-ray examination shows an opaque shadow in the mediastinum, and screening reveals the expansile pulsation of an aortic aneurysm, or the movement of retrosternal goitre during swallowing.
2. CT scan can exactly localize the site and origin of the tumour.
3. Bronchoscopy should always be carried out to exclude bronchogenic tumours.
4. Thoracoscopy may be carried out to show the outline of the tumour through the mediastinal pleura.

Treatment

- Radiotherapy is employed for enlarged malignant lymph nodes and malignant tumours, and may be supplemented with chemotherapy.
- Excision is indicated for cysts and simple tumours.

Thymic tumours

Tumours of the thymus are rare. They include thymoma (epithelioma), lymphomas, teratoma and lymphangioma. They may be symptomless, being discovered accidentally on X-ray examination, or may present clinically with the features of myasthenia gravis or the mediastinal syndrome.

Treatment is by radiotherapy followed by removal of the tumour through a sternum-splitting incision.

Cardiac Operations

Preoperative diagnostic procedures

- **ECG**
 - Resting. Rhythm, conduction abnormalities, chamber hypertrophy, established ischaemic changes and old myocardial infarction.
 - Exercise. Exercise-induced ischaemic changes.
- **Chest X-ray.** Cardiac enlargement and valve calcification.
- **Thallium isotope scan.** Areas of low isotope uptake indicate impaired myocardial perfusion.
- **Echocardiography.** Myocardial contractility, valve stenosis or regurgitation, and septal defects.
- **Cardiac catheter**
 - Chamber pressures. Assess left and right ventricular functions and assess valve diseases.
 - Angiography. Coronary artery anatomy.
 - O₂ saturation. Intracardiac shunts.
 - Cardiac output. Cardiac function and pulmonary vascular resistance. Left heart catheterization and coronary angiography are done by inserting a fine catheter into the brachial or femoral artery and threading it to the aorta and left side of the heart. Similarly right heart catheterization performed through a peripheral vein.

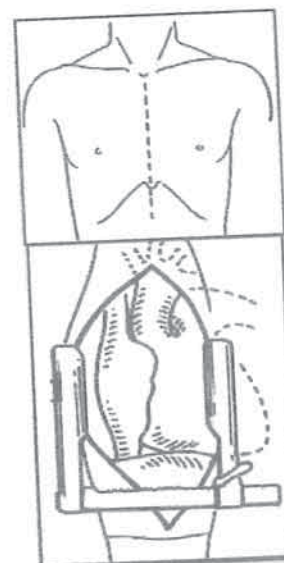


Fig. 28.26. Median sternotomy.

Operative procedure

Types of cardiac operations

1. Extracardiac operations are carried out on the main vessels or pericardium and present no more difficulties than operations on the lung. They include pericardiectomy, ligation of patent ductus, excision of coarctation and resection of aneurysms.

2. Closed intracardiac operations consist of blind procedures performed by the finger or by instruments inserted through the atrial or ventricular walls or through the base of one of the great vessels. They are designed mainly for the relief of stenosis of the mitral, aortic or pulmonary valves. These operations are infrequent in current cardiac surgery practice.
3. Open cardiac operations are performed under direct vision in a bloodless field within the chambers of the heart or great vessels. A median sternotomy is used (Fig. 28.26). To permit this type of operation, the venous inflow to the heart is diverted through an artificial heart lung machine (extracorporeal circulation).

Extracorporeal circulation (syn. cardiopulmonary bypass, open heart pump)

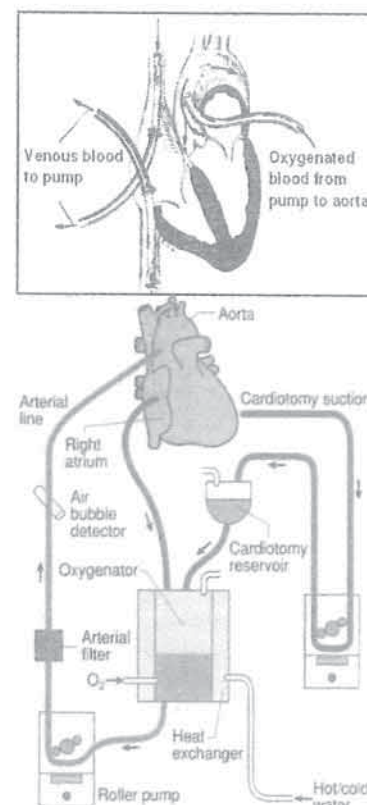
Extracorporeal circulation (Fig. 28.27) allows the surgeon to operate within the heart for long periods so that complicated lesions can be dealt with. The cardiopulmonary machine (pump) consists of

- Roller pumps which propel blood to and from the heart.
- An oxygenator which supplies oxygen and removes CO₂.
- Heat exchanger that control the blood temperature.

The machine is filled with fresh heparinized blood.

Venous blood is collected via cannulae inserted into the superior and inferior venae cavae.

Venous blood is pumped through the oxygenator and heat exchanger and is returned as arterial blood by a cannula into the femoral artery or the aorta.



Myocardial preservation

During the phase of extracorporeal circulation the heart is not perfused. The myocardium is usually preserved

by a combination of

1. **Cardioplegia.** The heart is induced to stop pulsations by infusing a cardioplegic solution in the coronary arteries. The main feature of this solution is its high potassium content. Cardioplegia minimizes myocardial oxygen requirement and allows the surgeon to operate at comfort on a still heart.
2. **Systemic hypothermia** down to 32°C.

Cardiac surgery complications

1. Complications of extracorporeal circulation
 - a. Cerebral ischaemia that may be severe and induces infarction or may be minimal and causes transient postoperative psychological disturbances or memory disturbances.
 - b. Activation of cytokines that induce systemic inflammatory response syndrome (SIRS, chapter 6). They are usually minor but in rare cases may cause coagulopathy and renal failure.
 - c. Haemolysis.
2. Low cardiac output.
3. Arrhythmias.

4. Fluid accumulation.
5. Pulmonary infection.
6. Mortality of cardiac surgery ranges from 2% for routine straightforward procedures to over 50% for complex emergency procedures.

Cardiac arrest

Cardiac arrest is sudden cessation of the pumping function of the heart.

Etiology: Two main causes are responsible

- (A) **Sudden severe reduction of the venous return to the heart may be due to**
- a. Sudden severe haemorrhage.
 - b. Failure of cardiac filling due to pericardiac tamponade, massive pulmonary embolism or tension pneumothorax.
 - c. Severe peripheral vasodilation which may occur after spinal anesthesia.
- (B) **Severe depression of the myocardial function due to**
- a. Myocardial infarction.
 - b. Hypoxia or hypercarbia.
 - c. Irritating drugs as epinephrine.
 - d. Traumatic myocardial contusions.
 - e. Disturbances of the electrolyte or acid base balance; e.g. acidosis or hyperkalaemia.
 - f. Hypothermia.

For simplification remember the common causes of cardiac arrest as the 5Hs and the 5Ts

- **5Hs** Hypovolaemia, Hypoxia, Hyperkalaemia, Hydrogen ion excess and Hypothermia
- **5Ts** Thrombosis of coronary arteries, Thromboembolism, Tension pneumothorax, cardiac Tamponade and Toxic disorders.

Cardiac arrest is classified into two main types according to the ECG findings

- (A) **Shockable rhythm** includes ventricular tachycardia or fibrillation.
- (B) **Non-shockable rhythm** includes cardiac asystole, and pulseless electrical activity. In the latter there is ECG rhythm compatible with a pulse, but the pulse is absent. The principal difference in the management of the two groups is the need for defibrillation in patients with shockable rhythm.

Diagnosis of Cardiac Arrest

The cardinal signs of cardiac arrest are

1. Absent carotid pulse.
2. Absent or gasping respiration (look, listen and feel for air).
3. Dilated pupils.
4. Under anesthesia ECG monitoring of the arrest and sudden cessation of bleeding are the main features.

N.B. The stethoscope has no place in the diagnosis of cardiac arrest. Once the condition is suspected the carotid vessels are palpable and if no pulse is felt, cardiac arrest must be assumed and resuscitation is started immediately.

Management of Cardiac Arrest

The brain can only tolerate 3 minutes of complete circulatory arrest, and unless the circulation is restored within this period, death will occur or permanent neurological damage will result if the patient survives. The proper management is carried out "immediately" and consists of three phases

Pre-hospital management [cardiopulmonary resuscitation (CPR)]

- External cardiopulmonary resuscitation by direct mouth-to-mouth breathing and closed cardiac massage is instituted at once.
- The aim is not to restart the heart but to provide an artificial circulation and artificial ventilation until appropriate facilities are available.
- **Technique**
 1. The patient is laid flat on a firm surface and the legs are raised to increase the venous return to the heart. The head is tilted back with one hand to ensure a clear airway, and the nostrils are closed with the other hand while the mouth is applied to the patient's mouth to fill his lungs with expired air at a rate of 10-15 times per minute (Fig. 28.28, 29).
 2. At the same time, a second person performs external cardiac massage by compressing the lower half of the sternum against the spine at a rate of about 100 times per minute (alternate 2 breaths and 30 sternal compressions). The person compressing the sternum stops while the other gives mouth-to-mouth breathing.
 3. The efficiency of ventilation and massage must be monitored by observing chest expansion and palpating the femoral pulse. The patient should then be transferred to hospital as soon as possible, with direct mouth-to-mouth breathing and cardiac massage continued until endotracheal intubation and medical treatment are available.

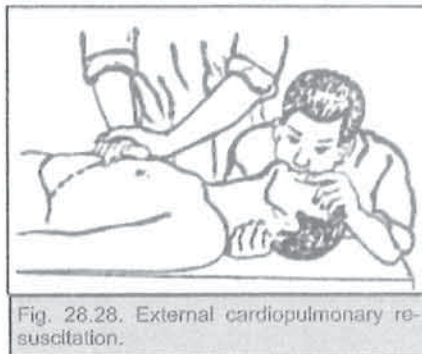


Fig. 28.28. External cardiopulmonary resuscitation.

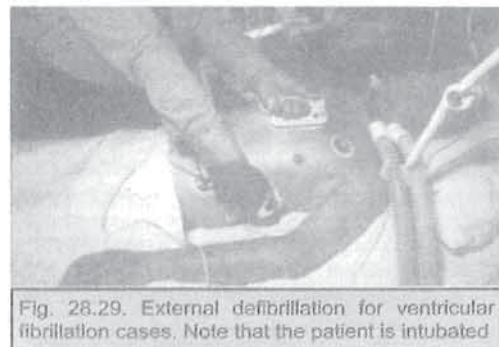


Fig. 28.29. External defibrillation for ventricular fibrillation cases. Note that the patient is intubated

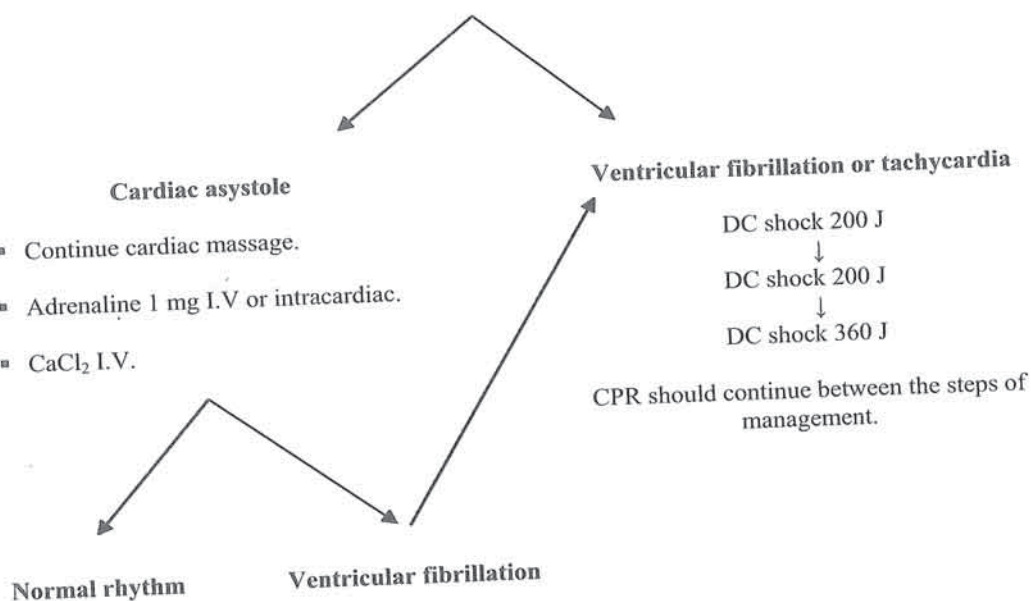
Hospital Management

- a. The aim is to restore the normal cardiac rhythm.
- b. An endotracheal tube is passed and the lungs are ventilated with oxygen.
- c. A venous cannula is inserted and suitable infusions are given.
- d. ECG monitoring is applied to differentiate cardiac asystole from ventricular fibrillation or tachycardia.

Open cardiac massage is employed if the chest is already opened or if closed massage has been unsuccessful. The chest is opened through the left fifth intercostal space and the pericardium is widely opened to permit bimanual massage with one hand

above and the other below the ventricular mass. Massage is continued at a rate of 60-80/minute until the cardiac tone returns and the heart becomes smaller, firmer and pinker.

The following is an algorithm for the management of cardiac arrest.



Subsequent treatment

Following restoration of normal rhythm the patient is kept under close observation in an ICU and the following measures are instituted to prevent and to treat re-arrest

- Blood pressure is kept over 90 mmHg by vasopressors and transfusion.
- If the chest has been opened it is left open for half an hour to watch for re-failure of the heart.
- Cardiac stimulants, e.g., noradrenaline, calcium chloride and digitalis, may be administered as needed.
- Hypothermia and dehydration therapy with mannitol, or concentrated glucose are induced if return of consciousness is delayed.
- Infusions of sodium bicarbonate solution are given to correct the metabolic acidosis produced by anoxia and blood transfusion.
- Hyperkalaemia is often present and should be treated with calcium chloride, glucose and insulin.
- Anuria and renal damage should be anticipated and prevented by adequate measures.
- The cause of cardiac arrest must be investigated and treated.

Key points in the management of cardiac arrest

- CPR should continue between the steps of management.
- The intervals between chest compressions and giving shock waves should not exceed 10 seconds.
- If there is doubt whether the rhythm is asystole or fine VF, do not attempt defibrillation; instead continue good quality CPR which may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation.

Principles of surgery for congenital heart diseases

Congenital heart diseases are, in general, surgically correctable. Surgery is done early after diagnosis. This is particularly important for cyanotic heart disease to prevent the development of irreversible lung changes that lead to pulmonary hypertension.

Patent ductus arteriosus

Treatment is by surgical closure that is best carried out between the ages of 3 and 5 years. Through a posterolateral thoracotomy the ductus is exposed and ligated using nonabsorbable material. There is no need for cardiopulmonary bypass.

Surgically correctable hypertension

- Renal diseases
 - a. Renal artery stenosis.
 - b. Unilateral pyelonephritis.
- Coarctation of aorta.
- Endocrine diseases
 - a. Pheochromocytoma.
 - b. Conn's syndrome.
 - c. Cushing's syndrome.

Coarctation of aorta

This is one of the causes of surgically-correctable hypertension.

Treatment

The operation is best carried out between the ages of 5 and 15 years.

- Excision and end-to-end anastomosis if possible.
- Excision and grafting with Dacron prosthesis if the constriction is a long one.

Tetralogy of Fallot

This is a complex anomaly consisting of four defects

1. Stenosis of the pulmonary artery.
2. Interventricular septal defect.
3. Dextroposition of the aorta which straddles the septal defect and receives blood from both ventricles.
4. Hypertrophy of the right ventricle.

Cyanosed is marked since birth, hence the term 'blue babies'.

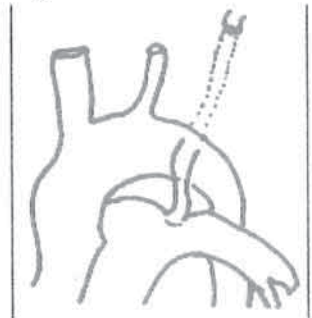


Fig 28 30 Blalock's operation

Treatment

1. Temporary palliative shunt operations aim at improving the blood flow to the lungs by means of a systemic pulmonary-artery anastomosis. In Blalock's operation (Fig. 2630), the subclavian artery is anastomosed to the pulmonary artery. The operation is done in the first year or two of life if the cyanosis is severe enough to make total correction with cardiopulmonary bypass a dangerous procedure.
2. Total correction is the definitive procedure. It requires cardiopulmonary bypass. The operation is usually advised before school age.

Atrial and ventricular septal defects

These diseases present either in children or adults. Surgery requires cardiopulmonary bypass but is usually easy with low risk of complications.

1. Small defects are closed directly by sutures.
2. Large defects are closed by Dacron grafts.

Principles of surgery for acquired valvular heart diseases

Surgery that preserves the patient's own valve produces better results than valve replacement.

Mitral stenosis

Indications for surgery

1. Progressive limitation of exercise tolerance.
2. Nocturnal dyspnoea.
3. Atrial fibrillation.
4. Congestive heart failure.
5. Mitral valve opening less than 1cm^2 as calculated by echocardiography.

Optimal timing

The ideal age is between 20 and 50, because below 20 reactivation of the disease may lead to recurrence, and over 50 myocardial ischaemia, atherosclerosis or emphysema may complicate the clinical picture.

Methods

1. Mitral valvotomy is the first choice if there is no associated incompetence and the valve leaflets are not calcified. Valvotomy may be a closed mitral commissurotomy, which does not require cardiopulmonary bypass, or open valvotomy.
2. Mitral valve replacement is needed for patients with mixed stenosis and incompetence and for calcified valves.

Mitral incompetence

1. If the valve is not heavily distorted or calcified, valve repair is preferred than replacement. The annulus of the valve is narrowed just enough to induce competence.
2. Mitral valve replacement is done in cases who have associated stenosis and those who have heavily distorted and calcified valves.

Aortic valve disease

Valve replacement on cardiopulmonary bypass is the ideal treatment. Associated coronary artery disease is treated at the same time.

Artificial valves (Fig. 28.31)

Types

1. **Prosthetic valves.** These have different designs, e.g., caged ball and tilting leaflets. Turbulent flow through these valves is likely to produce thrombosis. The patients should, therefore, be anticoagulated for life.
2. **Bio-prosthetic valves.** Valve leaflets are either porcine, bovine or human from fresh cadavers. The valve is suspended on a prosthetic ring to allow it to be sewn in place. The recipient does not need anticoagulation. The lifetime of such valves is shorter than prosthetic valves, but the avoidance of anticoagulation makes them the preferred choice in the elderly.

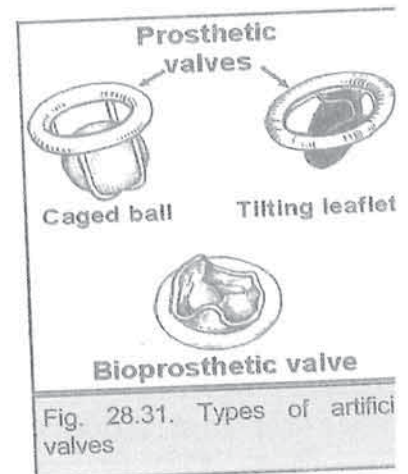


Fig. 28.31. Types of artificial valves

Complications of valve replacement

1. Prosthetic valve thrombosis if anticoagulants are discontinued.
2. Valve breakdown.
3. Complications of anticoagulants, particularly bleeding.
4. Valve infection, which is similar to bacterial endocarditis of native valves.

Ischaemic heart disease

Coronary anatomy

- There are two main coronary arteries; the right and the left coronaries.
- Both arise from the ascending aorta.
- The left coronary artery divides into two large branches; the left anterior descending (LAD) coronary which supplies the anterior wall of left ventricle and anterior two thirds of interventricular septum, and the circumflex (CX) coronary which supplies the posterior and lateral walls of left ventricle.
- The right coronary artery (RCA) supplies the right atrium and right ventricle directly and through its marginal branch.
- The RCA, LAD and CX arteries are each considered to be a "vessel system". Disease of any one of them or its branches is termed one vessel disease. Similarly, two and three vessel disease indicates involvement of two and three systems, respectively.

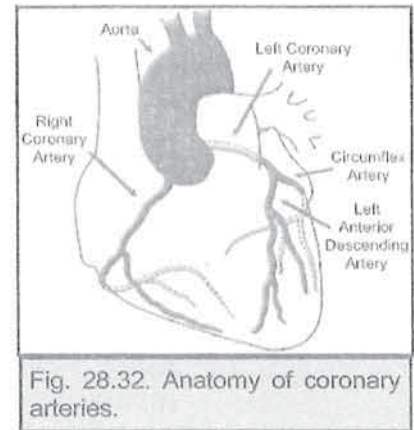


Fig. 28.32. Anatomy of coronary arteries.

Pathology and diagnosis

1. Cardiac ischaemia is due to narrowing of the coronary arteries by atherosclerosis.
2. The most common symptom is angina pectoris but the disease may culminate in coronary thrombosis with myocardial infarction, heart failure or ventricular fibrillation and sudden death.
3. The diagnosis is usually established by the history, ECG and exercise tolerance tests. Coronary arteriography (Fig. 28.33 & 28.34) is necessary before surgical treatment to determine the sites of occlusion, the patency of the vessels beyond the obstruction and the function of the ventricles.
4. In atherosclerosis, the basic lesion is a segmental atheromatous plaque, often localized within the first 5 cm of the origin of the coronary artery. Multiple areas of occlusion are common, and about 50% of patients with severe angina have involvement of all 3 major arteries, viz, anterior descending, circumflex, and right coronary.

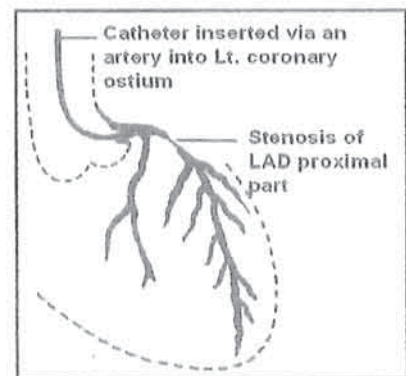


Fig. 28.33. Coronary angiography

Treatment

Medical

1. Moderation of activity
2. Control of risk factors. Prohibition of smoking and control of diabetes, hypertension and hyperlipidaemia.
3. Medications. Coronary vasodilators (nitrates), beta blockers and calcium channel blockers are effective in most cases.

Coronary revascularization

Indications

Severe ischaemia despite adequate conservative treatment

Methods

1. Percutaneous transluminal coronary angioplasty (PTCA) is done by inserting a balloon catheter inside the stenosed artery. The balloon is inflated to dilate the artery (Fig. 28.34). The dilated artery is usually supported by a stent to keep it open. PTCA is most useful for a short arterial narrowing.
2. Coronary artery bypass graft (CABG)

■ Indications

- Coronary arteries that are not suitable for PTCA.
- Stenosis of main stem of left coronary artery.
- Three-vessel disease, particularly in diabetics.
- Surgery for complications of infarction, which include mitral valve reflux due to rupture of papillary muscle, ventricular septal defect due to damage of the septum, and aneurysm of left ventricle due to its weakness. Operative correction of these complications is commonly associated with coronary bypass. The mortality is high in these cases.

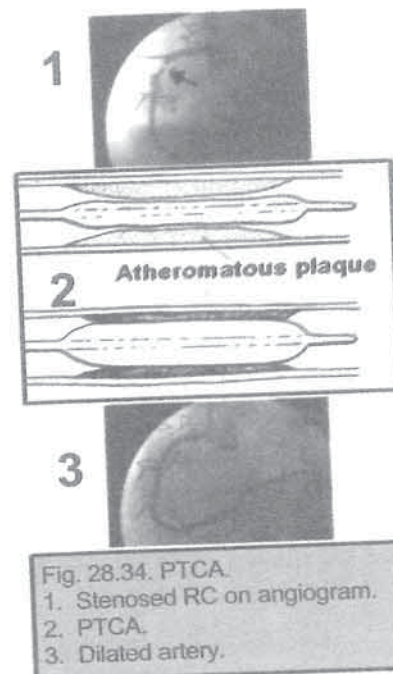


Fig. 28.34. PTCA.
1. Stenosed RC on angiogram.
2. PTCA.
3. Dilated artery.

■ Technique of CABG

- Open cardiac operation using extra-corporeal circulation and cardioplegia.
- Coronary bypass can be done by either:
 - Reversed saphenous vein grafts. The vein graft is anastomosed proximally to the ascending aorta and distally to the diseased coronary artery beyond the stenosis.
 - Internal mammary artery. It is usually employed as a pedicled graft, when it is left attached to the subclavian artery proximally, and is anastomosed to the diseased coronary artery distal to the stenosis. This graft has a better five and ten-year patency rates than saphenous vein grafts (Fig. 28.35).
 - A combination of both types is usually employed. The left internal mammary artery is used for a vessel at the front of the heart, and vein grafts are used to bridge other occlusions.
 - The use of the radial artery as a vascular graft is gaining recent popularity.

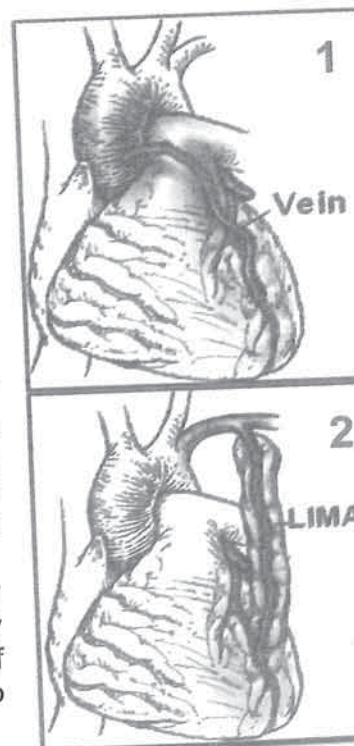


Fig. (28.35) CABG.
1. Saphenous vein grafts.
2. Left internal mammary artery (LIMA).

■ Results of CABG

- Angina is relieved completely in 70% of cases and is significantly improved in the remainder.

- Angina recurs with a frequency of about 10% per year.
- Straightforward CABG carries a mortality of 2-3% and a 1-2% risk of stroke.

Thoracic aortic aneurysms

- Aneurysmal dilatation may affect the aortic arch, the descending thoracic aorta or may extend into the abdomen forming a thoracoabdominal aortic aneurysm.
- The usual cause is atherosclerosis. Other causes include trauma and Marfan's syndrome. Syphilitic aneurysms are rarely seen nowadays.
- The aneurysm presents on account of its space-occupying manifestations, i.e., mediastinal syndrome. It is likely to compress superior vena cava, trachea, oesophagus and recurrent laryngeal nerve. It may also erode the vertebrae.
- CT scan or MRI is the investigation of choice.
- Surgery entails excision of the diseased segment and interposing a synthetic graft. The three branches of the arch are implanted in the graft.
- One possible complication of thoracic aortic surgery is paraplegia as a result of interrupting the blood supply of the spinal cord.

Aortic dissection

Predisposing factors Atherosclerosis and hypertension.

Pathology

- An intimal tear allows blood to dissect its way in the media in a caudad direction.
- Blood in the false lumen may rupture inside the true lumen or may rupture to the outside causing fatal haemorrhage. As blood dissects its way distally it occludes the ostia of aortic branches causing ischaemia. Of particular importance are the coronaries, the renal and the mesenteric arteries.
- There are two main types.
 - Type A. This is the more serious type. Dissection starts in the ascending aorta. It is likely to produce aortic incompetence, coronary artery occlusion and fatal cardiac tamponade.
 - Type B. Dissection starts distal to the arch.

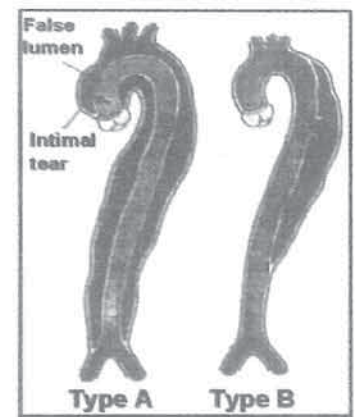


Fig. (28.36) Types of aortic dissection

Clinical features

Severe chest pain that radiates to the interscapular region. It is commonly misdiagnosed as acute myocardial infarction (MI). Actually MI may also be present in type A and diagnosis is not easy.

Investigations

- ECG
- CT
- Aortography
- Echocardiography; particularly the trans-oesophageal type.

Treatment

- Type A requires urgent surgery for the placement of a synthetic graft at the aortic root.
- Type B is treated by antihypertensives. Elective surgery may be needed in cases of chronic extension of dissection.

OESOPHAGUS

Anatomy

Course and relations

The oesophagus is a long muscular tube, 25 cm in length, extending from the pharynx at the level of the sixth cervical vertebra to the stomach in the abdomen (Fig. 29.1).

- In the neck it lies behind the trachea.
- In the mediastinum, it passes behind the left bronchus then behind the pericardial sac.
- It reaches the abdomen through the oesophageal hiatus which is formed mainly by the right diaphragmatic crus. The oesophageal opening in the diaphragm is one inch to the left of the midline at the level of the body of the tenth thoracic vertebra.
- An oesophageal segment of a variable length is present in the abdomen and this has a vital role in the lower oesophageal sphincter mechanism.

Wall layers

- Mucosa made of stratified squamous epithelium. At the cardia the epithelium is similar to that of the gastric mucosa but without peptic or oxyntic cells. The mucosal layer of the oesophagus is the strongest layer to hold sutures while performing an anastomosis.
- **Submucosa.**
- **Muscle layer.** The musculature of the upper third of the oesophagus is striated. This is gradually replaced by involuntary smooth muscle fibres arranged as inner circular and outer longitudinal layers.
- A layer of loose areolar tissue. There is no serosal lining.

Arterial supply

- The cervical oesophagus is supplied by the inferior thyroid arteries.
- The thoracic oesophagus is supplied by branches from the aorta and bronchial arteries.
- These oesophageal vessels are supplemented by branches arising from left gastric artery in the abdomen.

Venous drainage

There are subepithelial, submucous and perioesophageal venous plexuses from which blood drains to the inferior thyroid veins, azygos and hemiazygos veins and the left gastric vein. It is to be noticed that the venous

CHAPTER CONTENTS

- Anatomy
- Surgical physiology
- Oesophageal injuries
 - Corrosive & oesophageal injury
- Neuromuscular oesophageal abnormalities
- GORD
- Oesophageal hiatus hernia
- Carcinoma of the oesophagus
- Oesophageal replacement surgery
- Dysphagia

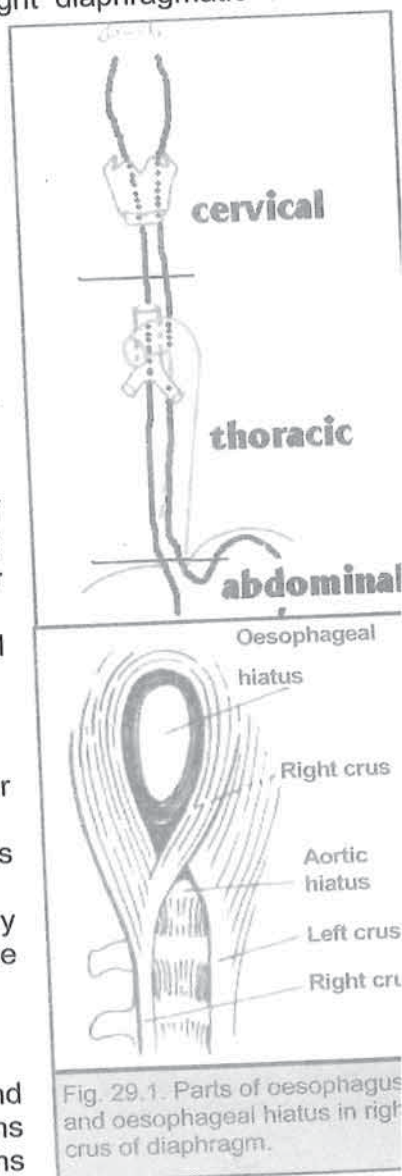


Fig. 29.1. Parts of oesophagus and oesophageal hiatus in right crus of diaphragm.

plexuses in the lower oesophagus represent a channel of communication between the tributaries of the portal circulation (left gastric vein) and those of the systemic circulation (azygos and hemiazygos veins).

Lymphatic drainage (Fig. 29.2)

Lymphatic vessels run longitudinally in the wall of the oesophagus and then penetrate the muscular layer to reach the lymph nodes.

- The cervical oesophagus drains into the cervical lymph nodes.
- The thoracic oesophagus drains to the tracheal, tracheobronchial and posterior mediastinal lymph nodes.
- The lower oesophagus drains to the left gastric and coeliac lymph nodes.

Innervation

- The cervical oesophagus is supplied by the recurrent laryngeal nerves.
- The thoracic oesophagus is supplied by the vagus nerves which control its peristaltic waves.
- The lower oesophageal sphincter has some degree of autonomy.

Physiological narrow areas

There are 3 points of physiologic narrowing of the oesophagus (Fig. 29.3)

1. The cricopharyngeus sphincter.
2. Behind the aortic arch and the left main stem bronchus.
3. At the oesophageal hiatus.

Surgical physiology

At either end of the oesophagus there is a strong sphincteric mechanism that allows one-way passage only except under unusual circumstances.

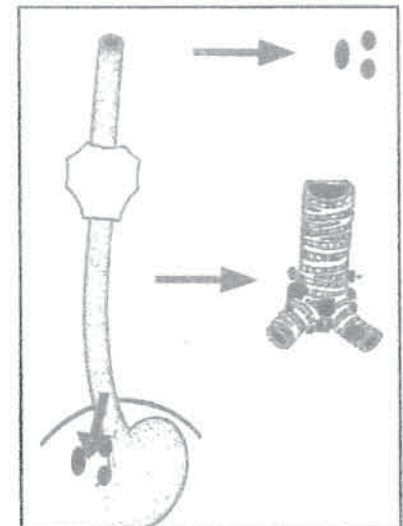
The upper oesophageal sphincter is formed of the cricopharyngeus muscle and a few centimeters of the upper cervical oesophagus.

The lower oesophageal sphincter is a physiological rather than an anatomical sphincter. It will be discussed in the paragraph of GORD.

Oesophageal injuries

Foreign bodies

Swallowed foreign bodies may be arrested in the oesophagus. If the foreign body is radio-opaque it will be visualized by a plain X-ray, otherwise a dilute barium swallow will reveal it. Foreign bodies can be removed by the rigid or the fiberoptic oesophagoscope.



• Fig 29.2. Lymphatic drainage of the oesophagus.

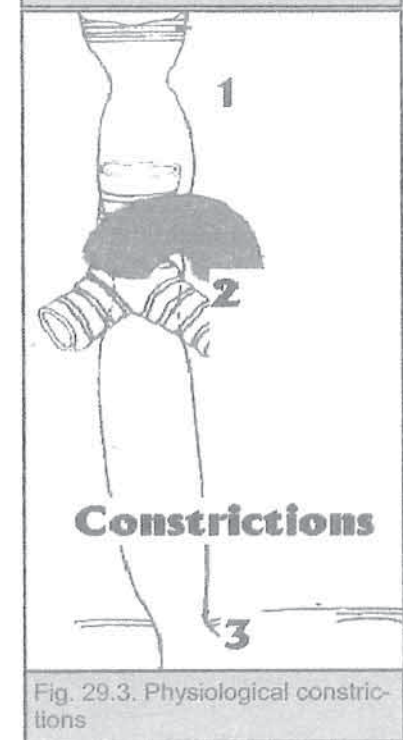


Fig. 29.3. Physiological constrictions

Oesophageal perforation

Aetiology

1. Traumatic

- a. Penetrating or blunt injuries to the neck or the chest.
- b. Instrumental. During oesophagoscopy or dilation of a stricture. The commonest site of injury following oesophagoscopy is at the level of the cricopharyngeus muscle.
- c. Foreign bodies.
- d. Swallowing of corrosives.

2. Pathological, e.g. carcinoma.

3. Spontaneous. The condition is likely to affect victims of head trauma and the drunken. In both situations there is vomiting and incoordinated oesophageal motility. The lower oesophagus fails to relax in front of the forcibly ejected vomitus. Pressure markedly rises in the lower part of the oesophagus, and its wall is stretched. As a result the wall gives way either partially where the mucosa only is split producing severe bleeding (Mallory-Weiss syndrome), or completely through the whole wall thickness. The tear is a longitudinal one in the lower part of the oesophagus, and is usually situated in its left posterior aspect.

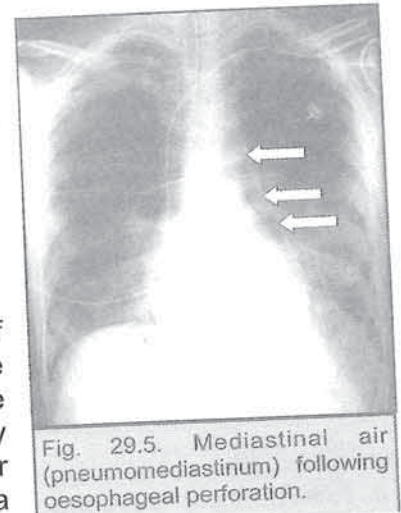


Fig. 29.5. Mediastinal air (pneumomediastinum) following oesophageal perforation.

Clinical picture

1. Severe pain at the site of rupture.
2. The patient is acutely ill with fever, tachycardia and hypotension.
3. Mediastinal emphysema appears as crepitus at the base of the neck, late subcutaneous emphysema extends over the chest wall and neck.
4. If the rupture penetrates the pleural cavity, pneumothorax, or pleural effusion occurs. This will lead to respiratory distress.

Investigations

- Plain chest x-ray reveals mediastinal emphysema (Fig. 29.5) or hydropneumothorax.
- Gastrografin swallow will reveal the site and extent of rupture.

Treatment

1. Cervical perforations

- a. Nil by mouth.
- b. Intravenous hyperalimentation.
- c. Drainage of the extravasated fluid,
- d. Intensive antibiotics.
- e. If the case is early, surgical closure of the perforation may be successful.

2. Thoracic Perforation

- a. If the diagnosis is early, suture of the perforation and chest drainage is often successful. A flap from the gastric wall may be utilized to close the perforation.
- b. If the diagnosis is delayed, any attempt to close the perforation will fail. Oesophagectomy and a gastric pull up operation with chest drainage may save the patient.

Corrosive oesophageal injury

Unfortunately many Egyptian children still fall victims to this tragedy.

Aetiology

Swallowing of corrosive acids, alkalies or household bleaches, the common example is potash.

Pathology

- The extent of damage caused by a caustic agent depends upon the concentration of the chemical agent and the duration of tissue contact. Corrosive acids produce coagulative necrosis that impedes deep penetration and so it is less injurious than alkalies.
- The corrosive injury is actually a chemical burn which may involve the oropharynx, larynx, oesophagus, or even the stomach. The burn may involve the superficial layers of the oesophagus or it may include the whole thickness with even perforation of the oesophagus.
- The injured wall of the oesophagus will be replaced by scar tissue leading to a stricture.

Treatment

Immediate treatment

1. Ask the patient to swallow some water or milk to dilute the effect of the corrosive. Gastric lavage is contraindicated.
2. Look for signs of laryngeal oedema as stridor or dyspnea. If present, introduce an endotracheal tube.
3. An analgesic is prescribed for the pain.
4. Oesophagoscopy is performed after 24 hours except in cases with laryngeal oedema or in patients in whom perforation is suspected. The aim of oesophagoscopy is to confirm the presence of the corrosive injury. The oesophagoscope should not be introduced through the damaged area, lest it would perforate the oesophagus.
5. It has been proved that early administration of corticosteroids and antibiotics after the corrosive burn markedly diminishes the incidence of oesophageal scarring. Corticosteroids should not be administered for more than 3 weeks.
6. A barium swallow will reveal the exact level and extent of the stricture. Sometimes, multiple strictures may be present (Figs. 29. 6).

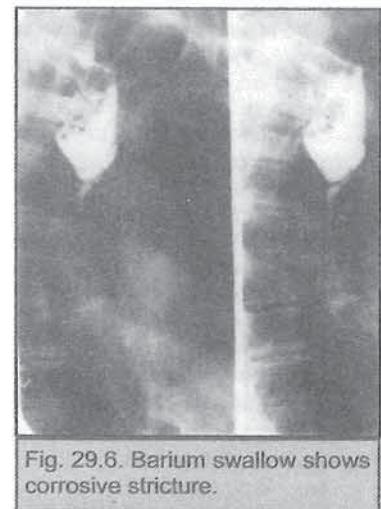


Fig. 29.6. Barium swallow shows corrosive stricture.

Later definitive treatment

Endoscopic dilatation

A course of gradual dilatation of the oesophagus by bougies is started. In young children this may need general anaesthesia. Dilatation should not be started before the end of the first week. The dilatation should be very gradual. Either mercury filled rubber bougies or hydrostatic polyurethane balloons can be used.

Criteria of success of dilatation treatment

1. Subjective improvement in swallowing.
2. Progressive increase in the diameter of the dilator which can negotiate the stricture.
3. The patient is puffing on weight.

Surgery

Indications. Surgery is indicated for extensive and persistent strictures which fail to dilate.

1. The need for frequent dilatations.
2. If dilatation is hazardous or difficult.

Usually these patients are in a poor nutritional status and cannot withstand major surgery. A gastrostomy operation is a minor procedure which can dramatically improve the patient's condition before definitive surgery.

Operation. Once the patient is fit, a colon bypass operation is performed. The idea of the operation is to isolate a segment of the colon (right or left colon) with intact blood supply. This segment is passed in a retrosternal tunnel and anastomosed to the cervical oesophagus above and to the anterior wall of the stomach below (Fig. 29.7). Some surgeons excise the strictured oesophagus as it is precancerous.

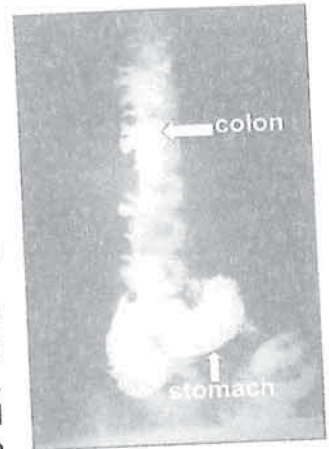


Fig. 29.7. Barium swallow shows colo bypass

Neuromuscular oesophageal abnormalities

Achalasia of the oesophagus

This is a functional disorder of the oesophagus that is characterized by two defects

1. Weak peristaltic waves in the body of the oesophagus.
2. Failure of the cardiac sphincter to relax during swallowing, the result is a functional obstruction with progressive dilatation of the oesophagus.

Aetiology

The aetiology is not exactly known but it is presumed to be due to degeneration of the vagal fibres and the ganglia in the Auerbach's plexus of the oesophagus itself. Another view considers the disease as an autoimmune disorder as there is infiltration of the oesophagus by T-lymphocytes.

Clinical picture

- The disease occurs more often in the second to the fourth decades and is equal in males and females.
- The main symptom is long-standing, slowly progressive, painless dysphagia which may be more to fluids than solids. At first the dysphagia is intermittent but becomes more constant as the disease progresses.
- Regurgitation of foul smelling fluid especially by night may be a prominent feature.
- The patient may have pulmonary symptoms as aspiration, wheezing and chronic cough.
- The general condition of the patient is usually reasonable contrary to patients with carcinoma of the oesophagus.

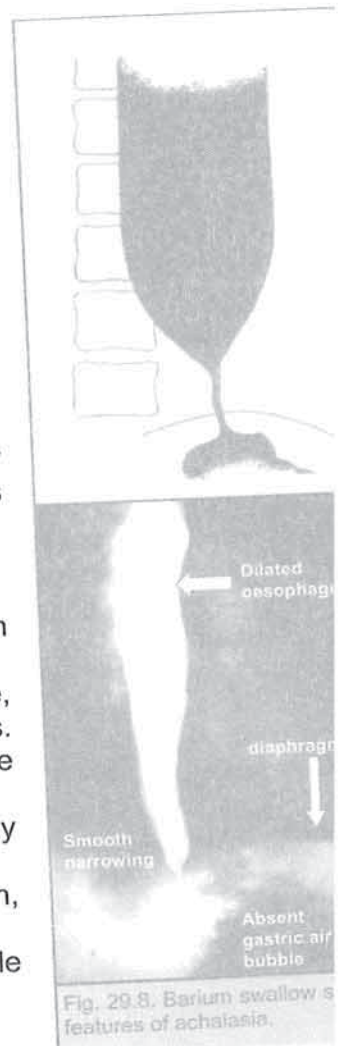


Fig. 29.8. Barium swallow features of achalasia.

Investigations

Plain chest x-ray may show a fluid level in the thorax, widening of the mediastinum or aspiration pneumonitis.

Barium swallow is the main diagnostic tool (Fig. 29.8).

1. In early cases there is only some hold-up of barium at the lower end of the oesophagus.
2. In advanced cases, moderate to huge dilatation of the oesophagus occurs, which may also show tortuosity (Figs. 29.9). The latter is called sigmoid oesophagus.
3. Smooth round termination of the lower end of the oesophagus (Hen's beak).
4. Absence of air in the fundus of the stomach due to continuous stagnation of fluids in the oesophagus.



Fig. (29.9) Markedly dilated tortuous esophagus

Oesophagoscopy

- It reveals a dilated oesophagus full of retained food and fluids. The cardia does not relax and may be eccentric in position.
- The main value of oesophagoscopy is to exclude the presence of carcinoma, which may complicate 5% of cases of achalasia after 20 years.

Manometric studies

- There are weak peristaltic waves in the body of the oesophagus after deglutition.
- Failure of relaxation of the cardia in response to swallowing efforts.

Treatment

- Drugs as nitrates or calcium channel blockers are of a very limited clinical value.
- Forceful dilatation of the cardia by hydrostatic or pneumatic dilators. The idea is to place a special balloon at the region of the cardia and then inflate the balloon to produce rupture of the circular muscle fibres to relieve the distal oesophageal obstruction (Fig. 29.10). The oesophagus is dilated to a diameter of 3 cm and the balloon is maintained for 1-5 minutes. Dilatation may be complicated by hemorrhage or perforation. 60% of patients remain well after one year, while only 30% remain symptomless after 5 years.
- **Cardiomyotomy (Heller's operation).** Surgery is the most reliable method of treatment. The operation can be performed via a thoracic or an abdominal approach. The principle is to expose the lower part of the oesophagus and cut the muscle fibres completely until the mucosa bulges through the incision. Some authors advise the addition of an antireflux procedure to prevent reflux oesophagitis following the myotomy (Fig. 29.10).

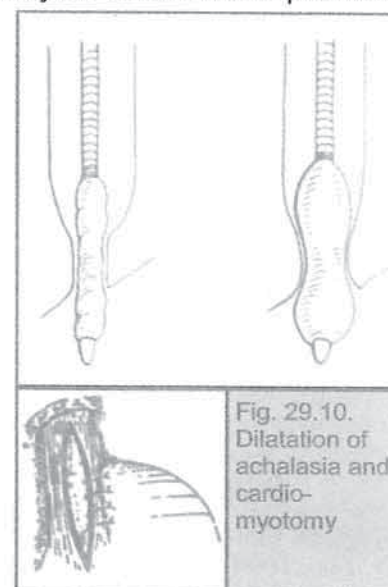


Fig. 29.10. Dilatation of achalasia and cardio-myotomy

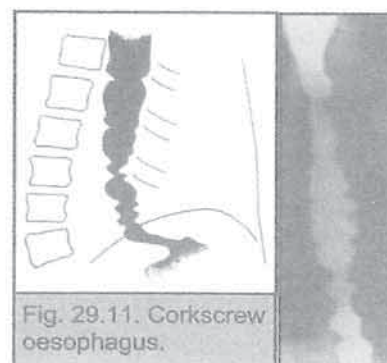


Fig. 29.11. Corkscrew oesophagus.

Recently, injection of botulinum toxin in the wall of the oesophagus at the spastic segment is tried.

Diffuse oesophageal spasm (corkscrew oesophagus)

In this condition the normal peristaltic waves are replaced by simultaneous, repetitive, and occasionally prolonged increases in pressure in response to deglutition (Fig. 29.11). The main symptom is retrosternal pain rather than dysphagia. Treatment is by long myotomy from the aortic arch to the cardia.

Gastro-oesophageal reflux disease (GORD)

Gastro-oesophageal reflux disease is the commonest upper digestive disorder with approximately 45% of the population having reflux symptoms. Peptic esophagitis is the commonest endoscopic diagnosis occurring in 25% of patients having an upper gastro-intestinal endoscopy.

Gastro- oesophageal reflux per se is a physiological phenomenon which occurs normally after meals. Up to 50 reflux episodes can occur every 24 hours, but most are very short lived. Exposure of the lower oesophageal mucosa to a pH less than 4 should not exceed 4% of the 24 hours period.

Gastro- oesophageal reflux in excess to this amount is pathological and leads to oesophageal mucosa sensitization.

Pathophysiology

Pathological GORD occurs when the balance between factors which promote reflux exceeds the effect of the factors which resist reflux and its damaging effect.

Mechanisms resisting reflux and damage include competence of the cardio-oesophageal junction and oesophageal clearance of the refluxate.

- (A) **Competence of the cardio-oesophageal junction** is due to the combined action of the lower oesophageal sphincter (LOS) which is rather physiological than anatomical and other factors (see below) maintaining a high pressure zone of 15-25 cm.H₂O
- The special arrangement of the gastric muscle fibers at the lower oesophagus; the clasps at the lesser curvature and the sling fibers of the greater curvature.
 - The 3 cm length of the intraabdominal esophagus creates a high pressure zone maintaining the competence of the lower oesophageal sphincter.
 - The action of the diaphragmatic crura especially the right crus which helps closure of the lower oesophagus during deep inspiration or during sudden rise of the intra-abdominal pressure as in sneezing and coughing.
 - The acute angle of His providing abrupt insertion of the oesophagus into the stomach maintaining a small diameter aperture of the distal oesophagus.
 - The mucosal rosette provides a valve like action at the lower oesophagus.

- (B) **Oesophageal clearance of the refluxate** from the lower oesophagus occurs by two mechanisms

- Volume clearance due to normal peristalsis during swallowing.
- Chemical clearance due to neutralization by swallowed saliva with its alkaline bicarbonate content.

These 2 mechanisms shorten the contact time between the refluxate and the oesophageal mucosa.

Factors promoting reflux and damage**(a) Dysfunction of cardio-oesophageal junction**

- a. Primary weakness of the LOS (hypotensive LOS).
- b. Short length of the intra-abdominal oesophagus (hiatus hernia).
- c. Abnormal high number of transient lower sphincter relaxations.

(b) Gastric distention impaired gastric emptying whether organic (gastric outlet obstruction) or functional (due to food which slows gastric emptying like fat, coffee and chocolate) may contribute to reflux.

Clinical Picture

1. **Typical symptoms** The typical symptoms of GORD are heart burn, regurgitation and dysphagia (typical triad). Other symptoms include water brush and odynophagia. Symptoms are aggravated by posture (lying flat, bending and stooping) and can be severe especially at night, after large meals.
2. **Atypical symptoms** Reflux may sometimes present with some atypical manifestations as:
 - Chest pain simulating coronary artery disease.
 - Pulmonary manifestations simulating bronchial asthma.
 - Laryngeal manifestations as persistent cough, change in the voice or choking episodes.
 - Rarely the patients may present with anemia or haematemesis in case of severe esophageal erosions.

Complications of GORD

1. **Reflux oesophagitis** Exposure of the lower oesophagus to the gastric acidity results in an inflammatory reaction with variable degrees of erosions and ulcerations. Less commonly, variable degrees of bleeding may occur.
2. **Formation of strictures and shortening of the oesophagus.**
3. **Barrett's oesophagus** it is columnar metaplasia of the stratified squamous epithelium of the lower oesophagus in response to the chronic acid irritation. The metaplasia may be of the gastric or intestinal type. Intestinal metaplasia is a premalignant condition which can develop into adenocarcinoma of the oesophagus.

Investigations

- **Upper GI endoscopy.** It can evaluate the degree of oesophagitis, diagnose the presence of hiatus hernia and a biopsy can be taken if suspicion of Barrett's oesophagus or malignancy is present.
- **Oesophageal manometry.** To test the pressure of the lower oesophageal sphincter.
- **pH study.** A small probe is passed through the nose to the lower oesophagus and positioned 5 cm above the LOS. The probe is connected to a digital recorder worn on a belt for 24 hours. The collected pH changes are then analyzed using a computer system. The patient is asked to record when he gets the symptoms of reflux oesophagitis. If the timing of these periods coincide with a low pH recording, this signifies that these symptoms are due to reflux.

Treatment**(A) Conservative**

Conservative treatment is used in the majority of cases as first line of treatment

1. Weight reduction will markedly improve the symptoms.
2. Stopping of smoking.
3. Avoidance of large, fatty, spicy and acidic meals, alcohol, chocolate and coffee.

4. Avoidance of recumbency after meals for at least 2 hours.
5. Elevation of the head and bed 15 degree to reduce reflux.
6. Medications Antacids, H₂ receptors blockers, proton pump inhibitors and prokinetic drugs.

(B) Surgical

Approximately 10-15% of patients with GORD will be referred for consideration to have anti reflux surgery. Indications of the surgery are

1. Failure of medical treatment.
2. Development of complications as severe oesophagitis persistent anaemia or oesophageal stricture.
3. Atypical symptoms (respiratory, pharyngeal and dental).
4. Non compliance of the patient.

Procedures

Anti reflux surgery

- Nissen fundoplication includes a complete wrap of the fundus of the stomach around the lower oesophagus (360°) to create a high pressure zone (Fig. 29.16). This operation can be done by open or laparoscopic surgery.

Oesophageal hiatus hernia

Hernias at the oesophageal hiatus may be sliding, paraoesophageal or mixed.

Sliding hiatus hernia

This is a condition in which the cardiac orifice and the adjoining part of the stomach herniate through the oesophageal hiatus into the posterior mediastinum (Fig. 29.12).

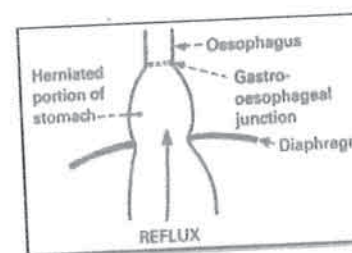


Fig. 29.12. Sliding hiatus hernia

Aetiology

1. Increased intra-abdominal pressure as in pregnancy, obesity or wearing of tight corsets.
2. Decreased elasticity of the right crus as in obesity and old age.

Pathology (Fig. 29.13)

1. The cardiac orifice and an adjoining part of the stomach herniate through the oesophageal hiatus into the posterior mediastinum. There is a small empty peritoneal sac on the left side of the stomach.
2. The oesophagogastric junction is no more in the abdomen, consequently, reflux of acid gastric juice to the lower oesophagus may occur leading to oesophagitis or even ulceration.
3. Reflex spasm of the oesophagus occurs and later even a fibrous stricture of the oesophagus occurs which will pull more and more stomach in the chest.
4. Prolonged oesophagitis will lead to replacement of the oesophageal mucosa columnar cells (Barrett's oesophagus) which is a precancerous condition.

The mere presence of a hiatus hernia on a barium meal study does not necessarily mean that the symptoms of the patient are due to it. Hiatus hernia may occur without reflux and there may be reflux without a demonstrable hiatus hernia.

Clinical picture

Age and sex hiatus hernia occurs more after the age of forty years and it is more common in females. The condition is commonly asymptomatic. If the episodes of reflux increase the patient will have the clinical picture of GORD

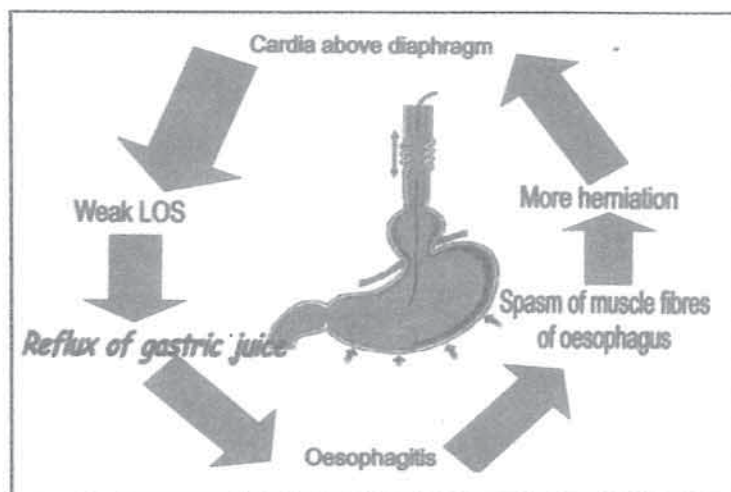


Fig. (29.13) Pathological sequelae of sliding hiatus hernia and gastro-oesophageal reflux

Investigations

1. Barium meal in the Trendlenburg's position
 - a. The hernia appears as a small epiphrenic bulge which is reducible in the erect position (Fig. 29.14).
 - b. Widening of the oesophageal hiatus.
 - c. Usually there is reflux of barium from the stomach to the oesophagus.
 - d. If there is oesophagitis, there will be irregularity of the oesophageal lumen with granular mucosal pattern.
2. Perform the investigations of GORD (Fig. 29.15).

Treatment

It should be stressed that sliding hiatus per se does not need treatment. Only if the symptoms or complications of GORD are severe, then surgery will be needed. The principles of the operation will be:

1. Reduction of the hernia.
2. Maintain of a segment of the oesophagus intra-abdominally.
3. Performance of an anti reflux mechanism (Nissen's fundoplication).
4. Repair of the right crass of the diaphragm.

Paraoesophageal (rolling) hernia

In this variety there is a true hernial sac into which the greater curvature of the stomach or even the entire stomach protrudes in front of the oesophagus in the posterior mediastinum. The oesophagogastric junction lies below the diaphragm (Fig. 29.17) and so reflux oesophagitis does not occur.



Fig. 29.14. Barium meal in head-down position shows a sliding hiatus hernia.

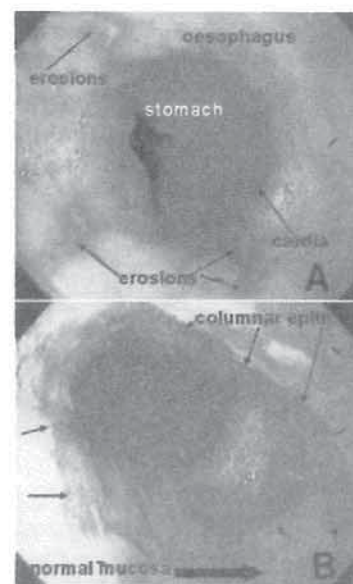


Fig. (29.15) Endoscopic findings in GORD. (A) Esophagitis and erosions. (B) Barrett's esophagus.

Clinical picture

1. Intermittent dysphagia.
2. Post prandial pain which may be confused with angia. The pain usually occurs rapidly after meals due to distension of the intrathoracic stomach.
3. Pressure on the heart may lead to cardiac symptoms.
4. Bouts of hiccough due to irritation of the phrenic nerve.
5. Occasionally strangulation of the herniated stomach occurs leading to pressure necrosis or even rupture. This will lead to mediastinitis or even empyema.

Investigations

Plain chest X-ray may show the gastric gas shadow in the chest. Barium meal will reveal the herniated stomach. The oesophagogastric junction is in its normal location (Fig. 29.18).

Treatment

Surgical interference is necessary to avoid fatal complications. Abdominal approach is recommended. The stomach is retracted downwards, the hernia sac is excised and the defect is closed.

Carcinoma of the oesophagus**Incidence**

This serious disease usually occurs after the age of 50 years and is more common in males than females except in the cervical oesophagus which in some areas may be more common in females.

Predisposing factors

1. Chronic irritation by certain diets. This may explain the high incidence in certain countries as China and Japan.
2. Excessive alcohol intake and smoking.
3. Corrosive stricture of the oesophagus.
4. Achalasia.
5. Long standing reflux oesophagitis.
6. Barrett's oesophagus which is lined by columnar mucosa. This is now supposed to be the most important predisposing factor.

Pathology**Distribution**

If the cardia is excluded, carcinoma is commonest in the middle third (45-50%) followed by the lower third (33%). The least site is the upper third and cervical oesophagus.

Gross appearance

As elsewhere in gastrointestinal tract the lesion may present as:



Fig. 29.16. Nissen's fundoplication.

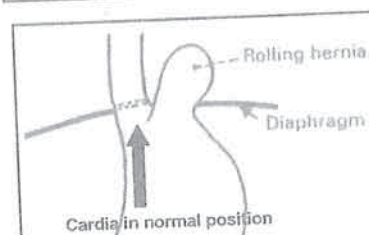


Fig. 29.17. Paraoesophageal hiatus hernia.

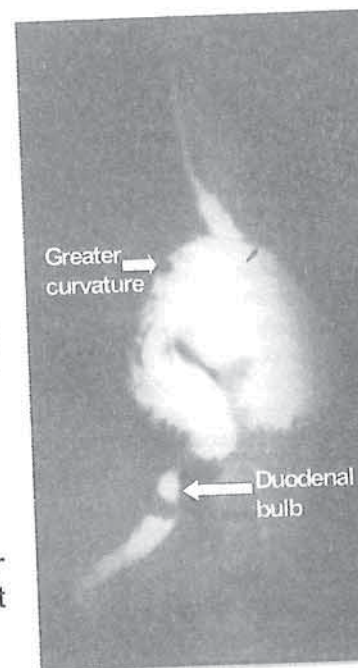


Fig. 29.18. Barium meal shows a paraoesophageal hernia this case the whole greater curvature of the stomach rolled up and herniated through the hiatus.

1. An annular schirrous lesion.
2. A malignant ulcer with raised everted edges.
3. Afungating cauliflower-like friable mass (Fig. 29.19).

Microscopic appearance

The most common is squamous cell carcinoma which may be well differentiated or less well differentiated with anaplastic features. Adenocarcinoma may occur in the lower oesophagus and is due to

1. Carcinoma occurring on top of columnar-cell lined lower oesophagus as in Barrett's oesophagus.
2. Upward spread of gastric carcinoma.
3. Recently, there has been an increase in the incidence of adenocarcinoma.

Spread

1. Direct spread. The lesion spreads in a circumferential and longitudinal directions in the wall of the oesophagus. Malignant cells can be detected in the submucosa many centimeters beyond the palpable edge of the growth. Finally the cells penetrate the muscle layer and the tumour may infiltrate adjacent structures as the trachea, pericardium, pleura, recurrent laryngeal nerves, the diaphragm, or the stomach.
2. Lymphatic spread. According to the site of the lesion, metastases to the draining lymph nodes may occur.
3. Blood spread. This is rather late. Lesions may be found in the lungs, liver, ribs or vertebrae.

Clinical picture

1. **Dysphagia.** Progressive continuous dysphagia is the main symptom, at first for solids then for soft diet and lastly for fluids. In advanced cases the patient cannot swallow his own saliva leading to continuous drooping of saliva and aspiration pneumonitis. By the time the patient complains of dysphagia, about 2/3 of the circumference of the oesophagus are involved by the tumour.
2. Regurgitation leading to pulmonary problems.
3. Severe and progressive loss of weight. In advanced cases the patient is very emaciated.
4. Haematemesis is not a common symptom.
5. In advanced cases the patient may complain of symptoms due to infiltration of adjacent structures as change of voice (recurrent laryngeal nerve) or recurrent choking due a tracheo-oesophageal fistula.
6. On examination signs of dissemination as pleural effusion, ascites, lymph nodes enlargement, hepatomegaly, and jaundice should be looked for.

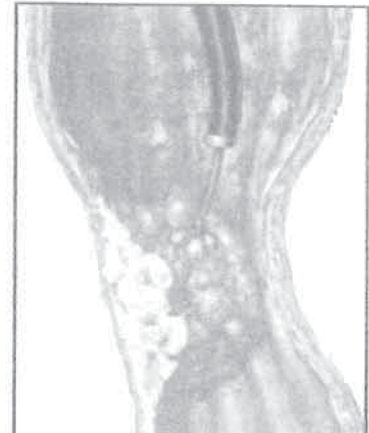


Fig. 29.19. Gross appearance of carcinoma of oesophagus.

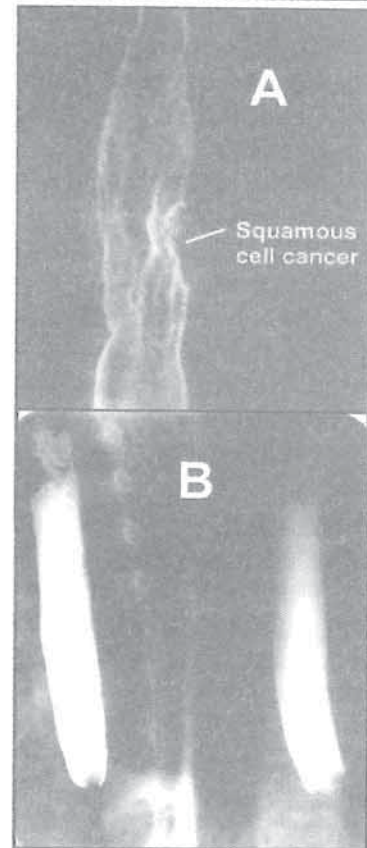


Fig. (29.20) Barium swallow of esophageal cancer. (A) Early case with irregular filling defect. (B) Almost complete occlusion of lumen with characteristic shouldering.

Investigations

■ Barium swallow

- Cauliflower like lesions will produce persistent irregular filling defects (Fig. 29.20A). Schirrous lesions will show an irregular narrowed segment, with overhanging margins. There is sudden transition between the dilated segment and the narrowed one leading to the characteristic shouldering (Figs. 29.20B). Peristaltic waves may be absent above the lesion due to submucosal infiltration of the oesophageal wall.

- Barium swallow can usually differentiate between achalasia and carcinoma of the oesophagus. In achalasia the lesion is at the level of the cardia with smooth rounded termination and with moderate or even huge dilatation of the oesophagus.

- **Oesophagoscopy** will demonstrate the lesion (Fig. 29.21) and multiple biopsies are taken.
- **Bronchoscopy** is advisable in lesions of the upper oesophagus to detect infiltration of the trachea on bronchi.
- **Plain chest X-ray** to detect pulmonary deposits or malignant effusion.
- **Abdominal ultrasound** may reveal liver deposits or ascites.
- **Endoluminal ultrasonography** can detect invasion of the mediastinum and lymph node enlargement.
- **CT scan** can demonstrate the extent of the lesion, the presence of lymph node deposits and the presence of infiltration of adjacent structures.

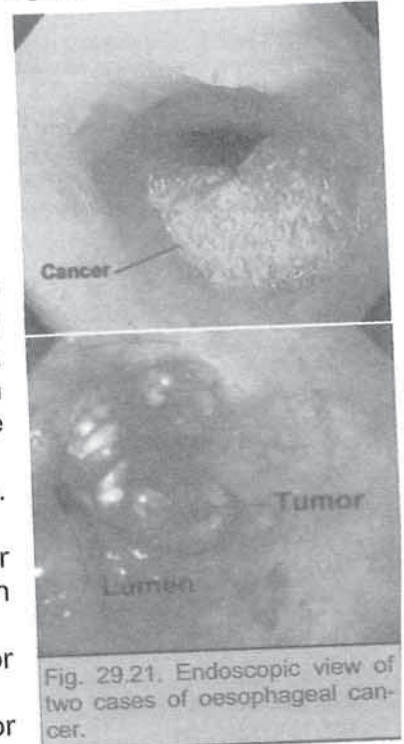


Fig. 29.21. Endoscopic view of two cases of oesophageal cancer.

Treatment

Palliative treatment

Unfortunately many of the patients with oesophageal carcinoma are diagnosed when the lesion has reached an advanced stage, and the chance of cure is very low. Before embarking on a major surgery for a patient with oesophageal cancer, thorough investigations should be performed to exclude those patients who are inoperable or have irresectable lesions.

Indications for palliative treatment

1. Patients with a poor cardio-respiratory status.
2. Patients with distant metastases as pulmonary deposits, liver metastases, supraclavicular glands and ascites.
3. Patients with advanced local spread as those with a tracheoesophageal fistula, recurrent laryngeal paralysis, infiltration of the pleura or pericardium or extensive lymph node deposits.

Aim of palliative treatment is to relieve dysphagia, i.e., to allow the patient to swallow.

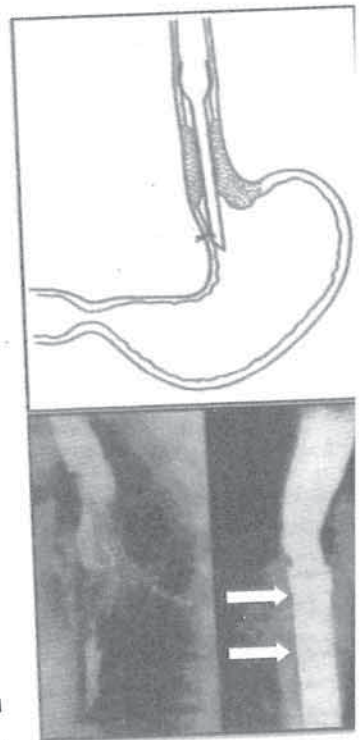


Fig. (29.22) Intubation restores patency of the lumen (arrows).

Methods of palliation

1. **Radiotherapy.** A dose of 4000 to 4500 rads, over a period of 4 weeks. This is particularly suitable for carcinomas of the upper oesophagus. Complications of radiotherapy include haemorrhage, perforation and pneumonitis.
2. **Intubation.** The idea is to insert a rigid tube through the stenosed segment, to keep a patent lumen (Fig. 29.22). There are tubes which are pushed through the lesion by the oesophagoscope, and there are those which are pulled from below after the performance of a gastrostomy. Examples of tubes include the Souttar and Celestin tubes. There are now also self-expanding tubes.
3. **Laser photocoagulation (Nd:YAG).** Laser energy causes tissue coagulative necrosis. Complications include chest pain and possible perforation.
4. **Gastrostomy.** This is performed when there is no other alternative. It does not relieve the patient from the inability to swallow his own saliva.

Radical surgery

Indications. Patients in a good general condition with a resectable lesion are offered the chance of major surgery.

The **idea of the operation** is to resect the lesion with an adequate safety margin on either side (10 cms) and then to restore the continuity of the gastrointestinal tract.

Types of operations. There are many approaches to perform these operations.

- For tumours of the lower third of the oesophagus, a left thoracoabdominal incision can be used and after excision of the oesophagus and the upper portion of the stomach, an oesophagogastric anastomosis is performed (Fig. 29.23).
- For tumours of the middle third, the stomach is mobilized through a midline incision, then the thorax is opened through the right 5th intercostal space and the oesophagus is excised down to the cardia. Then an oesophagogastric anastomosis is performed. This approach is called Ivor Lewis.
- Nowadays, many surgeons prefer to do total oesophagectomy through a transhiatal approach (without a thoracotomy) and then perform an anastomosis in the neck between the cervical oesophagus and the mobilized stomach (Fig. 2b.24).

Advantages of this operation

1. Guarantee of an adequate safety margin.
2. The stomach can be mobilized easily.
3. The oesophago-gastric anastomosis is performed in the neck. This is technically easy and should leakage occurs, it will be in the neck and does not lead to mediastinitis or empyema.
4. No need for a thoracotomy.

Oesophageal replacement surgery

1. **Gastric pull-up procedure** (Fig. 29.23). The idea is to mobilize the stomach completely with preservation of the right gastric and right gastroepiploic vessels. The stomach can be brought up to the neck. **Advantages of gastric replacement:**

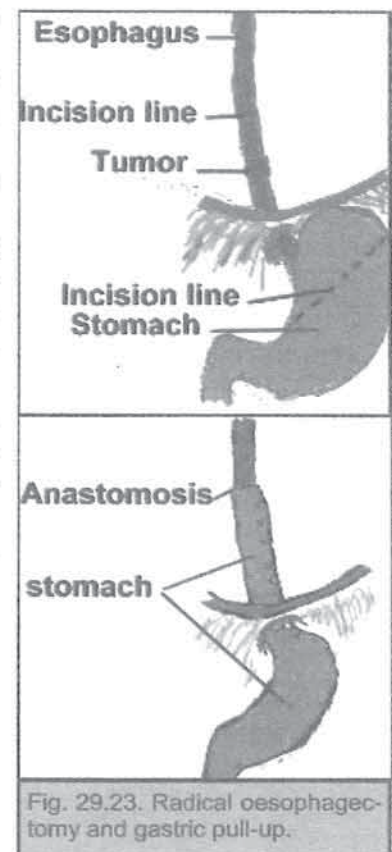


Fig. 29.23. Radical oesophagectomy and gastric pull-up.

- a. The stomach has a very rich blood supply.
 - b. Complete serosal covering.
 - c. Easy mobilization.
2. **Colon replacement.** A segment of the right or left colon is mobilized with preservation of the feeding artery and vein and the marginal artery near the wall of the colon. The colon is anastomosed below to the stomach and above to the cervical oesophagus (Fig. 29.25).
3. **Pectoralis major myocutaneous flap.** This can be used to replace a localized segment of the cervical oesophagus. The skin segment is rolled upon itself to form a tube with the epidermal surface facing inwards so that it lines the lumen.
4. **Free jejunal replacement with microvascular anastomosis.** A segment of jejunum is separated with preservation of the feeding artery and vein. Using microvascular techniques the feeding artery is anastomosed to an artery in the neck, e.g. the inferior thyroid artery and the feeding vein is anastomosed to one of the veins in the neck.

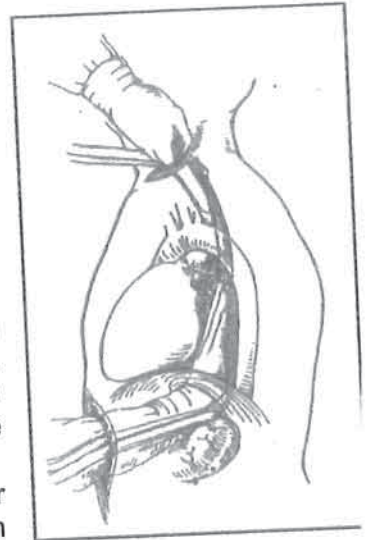


Fig. (29.24) Trans-hiatal approach to esophagectomy. Cervical and abdominal incisions only without a thoracotomy.

Dysphagia

Dysphagia means difficulty in swallowing. Odynophagia means painful swallowing.

Causes of dysphagia

Causes in the mouth Stomatitis-glossitis-neoplasms of the tongue and cheek.

Causes in the pharynx

1. Pharyngitis.
2. Retropharyngeal abscess.
3. Plummer vinson syndrome.
4. Pharyngeal diverticulum.
5. Post-cricoid carcinoma.
6. Neurpmuscular D.M. - Poliomyelitis - Myopathy - C.V.A.

Causes in the oesophagus

Mechanical

1. Causes in the lumen foreign bodies.
2. Causes in the wall
 - a. Congenital.
 - b. Traumatic as corrosive or post-operative stricture.
 - c. Inflammatory as in reflux oesophagitis.
 - d. Neoplastic as carcinoma.
3. Compression from outside. Malignant thyroid, malignant lymph nodes, aneurysm and mediastinal tumours.

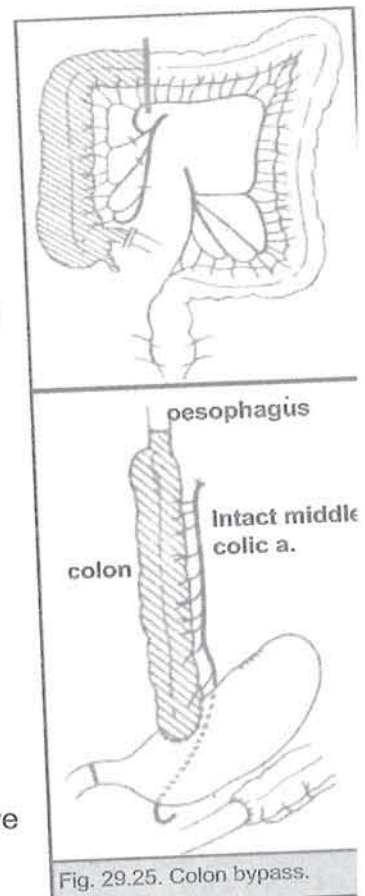


Fig. 29.25. Colon bypass.

Neuromuscular

1. Achalasia of the cardia.
2. Paralysis of the glossopharyngeal or vagus nerves.
3. Tetanus.
4. Myasthenia gravis.
5. Hysteria

Investigations

- Chest x-ray.
- Barium swallow.
- Pharyngoscopy and oesophagoscopy.
- C.T scan of the chest.

Points to Remember**Therapeutic uses of oesophago-gastroduodenoscopy**

1. Band ligation or sclerotherapy of oesophageal varices.
2. Control of bleeding from peptic ulcers by submucous adrenaline injections, laser or hemo-clips. Bleeding superficial gastric erosions can be controlled by the argon beam coagulator.
3. Percutaneous endoscopic gastrostomy (PEG).
4. Dilatation of benign oesophageal strictures and/or achalasia as well as dilatation of pyloric obstruction.
5. Stenting of malignant strictures of the oesophagus and trachea-oesophageal fistulae.
6. Recently, areas of oesophageal dysplasia or Barrett's oesophagus can be ablated superficially by endoscopic mucosal resection (EMR) or photodynamic therapy.
7. Removal of swallowed foreign bodies.
8. ERCP with removal of CBD stones, dilatation or stenting of biliary strictures.

Endoscopic grading of reflux oesophagitis

- Grade I hyperaemic mucosa.
- Grade II intermittent superficial ulcers.
- Grade III extensive ulceration.
- Grade IV stricture or Barrett's oesophagus.

Barrett's oesophagus

- 10% of the cases of GORD.
- Metaplasia of the lower oesophageal epithelium into columnar (intestinal type) epithelium.
- A precursor for ulcers, dysplasia, cancer in situ and adenocarcinoma.
- Regular endoscopic monitoring with multiple biopsies is essential.
- Endoscopic mucosa resection (EMR) using photodynamic therapy or argon beam coagulation may be used before progression to cancer.

STOMACH AND DUODENUM

Surgical anatomy

Parts of the stomach

The stomach is anatomically divided into (Fig. 30.1)

1. Cardia which lies immediately below the entrance of the oesophagus. It represents the upper end of the organ, but is not its highest point.
2. Fundus is that part of the stomach which lies above and to the left of the cardia.
3. Body which extends from the fundus to the antrum. The junction is a line drawn from the incisura angularis to meet the greater curvature.
4. Antrum is the distal part of the stomach which ends at the pyloric sphincter.

Functional division. From a functional point of view, the important division is between the antrum and the body.

1. The body secretes HCl, pepsinogen, mucus, and intrinsic factor.
2. The antrum secretes mucus and the hormone gastrin.

Therefore, the antral mucosa is alkaline while the rest of the stomach is acidic, a fact to remember when the sites of peptic ulcer will be discussed.

Layers of the stomach wall

1. Mucosa is formed of surface epithelium and an underlying thin sheet of muscle, the muscularis mucosa. The lining epithelium is formed of tall columnar cells and is pitted by the openings of the gastric glands. The glands are of two main types (Fig. 30.2)
 - In the proximal three fourths of the stomach (the parietal cell area), the glands contain oxyntic (parietal) cells that secrete hydrochloric acid and intrinsic factor, chief cells that secrete pepsinogen, mucous cells, and argentaffin cells.
 - In the distal fourth (the antral area), the parietal and chief cells are absent. G cells that secrete gastrin are found in this area. They are part of the APUD system of endocrine cells.
2. Submucosa.
3. Muscularis propria that in addition to the usual inner circular and outer longitudinal, has an innermost incomplete oblique layer.
4. Serosa.

CHAPTER CONTENTS

- Surgical anatomy
- Surgical physiology
- Acute gastric dilatation
- Peptic ulcer disease (general considerations)
- Acute peptic ulcer
- Chronic duodenal ulcer
- Chronic gastric ulcer
- Complications of peptic ulcer
- Complications of gastric operations
- Neoplasms of the stomach
- Gastrectomy
- Bariatric surgery

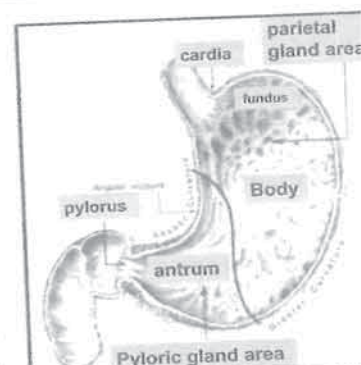


Fig. 30.1. Parts of the stomach

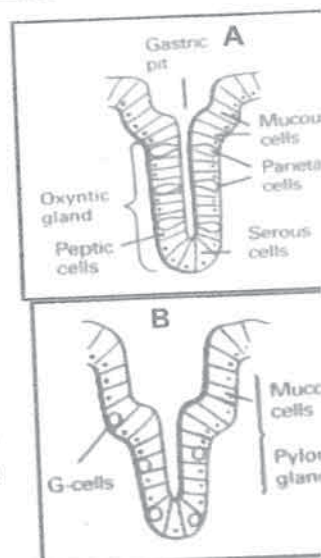


Fig. 30.2. Gastric glands. Body A. body B. antrum

Blood supply of the stomach

The stomach has a very rich arterial blood supply that is derived from the coeliac axis, being a part of the foregut (Fig. 30.3). There is a very rich anastomotic network in the submucosa so that it is difficult to tell which artery supplies which part of the stomach. This fact is surgically important for more than one artery can be ligated with no ill effects.

1. The left gastric artery is one of the three main branches of the coeliac trunk.
2. The right gastric artery is a branch of the hepatic artery. Both gastric arteries run on and supply the lesser curvature.
3. The left gastroepiploic artery is a branch of the splenic artery.
4. The right gastroepiploic artery is a branch of the gastroduodenal (from the hepatic artery). Both gastroepiploic arteries form a complete arch that runs on and supplies the greater curvature.
5. The short gastric arteries (vasa brevia) arise from the splenic artery and supply the fundus via the gastrosplenic ligament.

Corresponding veins finally end in the portal vein. The left gastric vein also has multiple anastomoses with the lower oesophageal venous plexus. These drain systemically into the azygos vein.

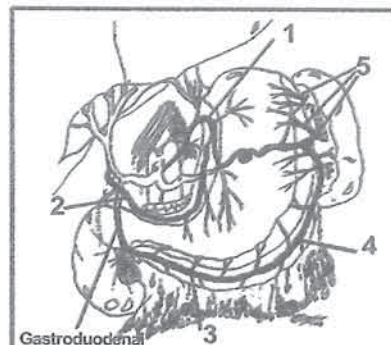


Fig. 30.3. Arterial supply of the stomach.

1. Left gastric a.
2. Right gastric a.
3. Right gastroepiploic a.
4. Left gastroepiploic a.
5. Short gastric arteries.

Nerve supply of the stomach

Parasympathetic nerve supply (Fig. 30.4) reaches the stomach via the anterior and posterior vagus nerves which enter the abdomen through the oesophageal hiatus of the diaphragm in close relation to the oesophagus. Each nerve trunk gives off a branch before continuing parallel to the lesser curve as the nerve of Latarjet that ends in the pylorus. The branch of the anterior vagus (hepatic branch) goes to the liver, while that of the posterior vagus goes to the coeliac plexus (coeliac branch) to share in the innervation of the gut to the mid transverse colon.

The main trunks as well as the nerves of Latarjet provide the stomach with preganglionic fibres that relay in ganglia within the stomach wall. The vagi contribute to acid secretion and to gastric motility.

Sympathetic nerve supply is from the greater splanchnic nerves, then the coeliac ganglion. Post-ganglionic fibres reach the stomach along its arteries. Afferent fibres carry visceral pain sensation.

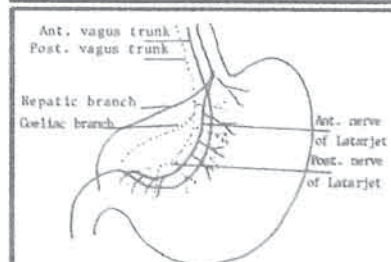


Fig. 30.4. Parasympathetic innervation of the stomach.

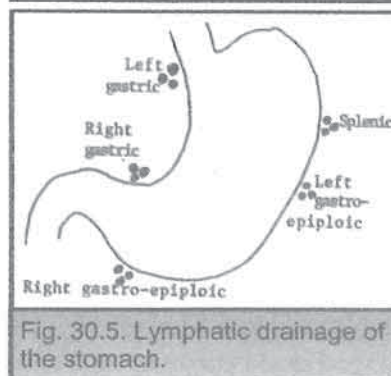


Fig. 30.5. Lymphatic drainage of the stomach.

Lymphatic drainage of the stomach

A sound knowledge of the lymphatic drainage of the stomach is essential for the proper treatment of its malignancies.

Primary stations (Fig. 30.5).

- The proximal half of the stomach drains to the left gastric lymph nodes and to the splenic lymph nodes.

- The antrum drains into the right gastric lymph nodes and to the subpyloric lymph nodes.
 - The greater curvature drains into lymph nodes along the gastroepiploic arch.
- These lymph nodes are the first tier lymph nodes.

Secondary stations

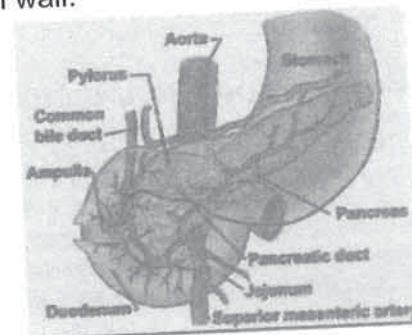
From the previously-mentioned groups lymphatics converge more proximally to end in the coeliac lymph nodes or the superior mesenteric lymph nodes. It must be realized, however, that lymphatics communicate freely in the stomach wall.

Anatomy of the duodenum

The duodenum is the first part of the small intestine. It roughly resembles the letter "C" that houses the head of the pancreas in its concavity (Fig. 30.6). It is divided into four parts, all except the first inch of its first part, are retroperitoneal.

- The first part is the horizontal portion distal to the pylorus. It is approximately two inches in length. It is related posteriorly to the common bile duct and the gastroduodenal artery.
- The second or descending part is about three inches. The common bile and the main pancreatic ducts commonly join and form a dilated area (the ampulla of Vater) before opening on the major duodenal papilla in the middle of the medial aspect of the second part of the duodenum.
- The third or transverse part is about four inches long. It runs horizontally to the left and is crossed anteriorly by the superior mesenteric vessels.
- The fourth part runs superiorly and to the left for one inch. At its terminal portion, the duodenojejunal (DJ) flexure, it changes direction abruptly and becomes the jejunum. This area of the duodenum is fixed in position by the ligament of Treitz.

The duodenal papilla in the second part marks the junction of the area derived from the foregut proximally, from that derived from the midgut distally. The former is supplied by the coeliac, and the latter by the superior mesenteric artery.



30.6. Anatomy of the duodenum

Blood supply

- The proximal half is supplied by the superior pancreaticoduodenal artery which is a branch of the gastroduodenal.
- The distal half is supplied by the inferior pancreaticoduodenal artery which is a branch of the superior mesenteric artery.

Surgical physiology

Functions of the stomach

1. Grinding and mixing of food into a homogenous chyme.
2. Food reservoir that allows controlled slow passage of chyme into the duodenum.
3. Early stages of protein digestion by pepsin.
4. Secretion of hydrochloric acid which
 - Activates the proteolytic enzyme pepsin.
 - Kills bacteria in swallowed food.
 - Facilitates iron absorption.
5. Secretion of intrinsic factor which is essential for absorption of vitamin B12.

Motility

1. Complimentary to the storage function is the process of receptive relaxation in which the body and fundus of the stomach relax to accommodate increasing volumes of food without major pressure increase.
2. Gastric motility is a function of the intrinsic nerve plexus, but is modified by the autonomic innervation and the content of food.
 - The parasympathetic system encourages gastric emptying. Division of the two vagal trunks induces gastric stasis, and also abolishes receptive relaxation.
 - The sympathetic system inhibits motility.
 - Solid, fatty, and hyperosmolar foods tend to retard gastric emptying.

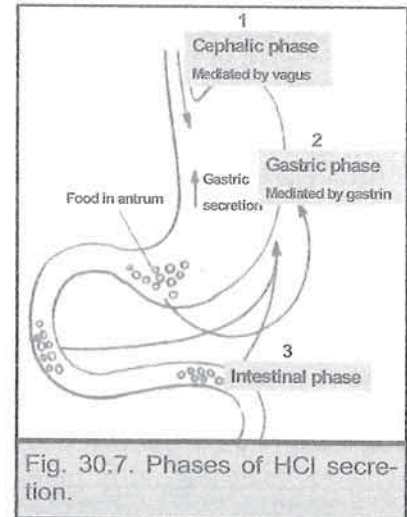


Fig. 30.7. Phases of HCl secretion.

Hydrochloric acid secretion

Gastric secretion in response to eating is described in three phases (Fig. 30.7).

1. Cephalic phase is mediated by vagal stimulation. It is provoked by the sight, smell, or thought of food.
 - Vagal stimulation has a direct effect on parietal cells, inducing acid secretion.
 - Stimulation of the vagi also induces gastrin release from the G cells of the antrum.
2. Gastric phase is mediated by the hormone gastrin. Gastrin is the most powerful stimulator of gastric acid secretion. It is released from the antral mucosa into the blood stream in response to mechanical distension of the antrum by food, and by the products of early protein digestion. Low pH less than 2, i.e. a high acidity inhibits gastrin release (a negative feed back mechanism). It seems likely that gastrin acts on the parietal cells through the release of histamine.
3. Intestinal phase offers the least contribution. The involved factors are poorly understood.

Gastroduodenal mucosal barrier

This barrier is formed of a group of factors that normally protect the mucosa from being attacked and digested by acid and pepsin. These factors are:

1. The mucus secreted by the mucosa serves as a protective lining.
2. Bicarbonate secretion by the mucosa which is mostly present between it and the protecting layer made of mucus. The surface pH is thus higher than the luminal pH.
3. Rapid regeneration of mucosal cells.
4. Abundant mucosal blood flow.
5. Prostaglandins which maintain the blood supply.

Factors that weaken the gastroduodenal mucosal barrier

1. Drugs particularly aspirin and non-steroidal anti-inflammatory drugs as they have anti-prostaglandin effect.
2. Duodeno-gastric reflux of bile.
3. Helicobacter pylori infection these organisms secrete urease enzyme leading to the liberation of ammonia which increases gastrin production. They also secrete proteases which destroy the mucosa.

A **balance** is normally present between the attacking forces (acid and pepsin) on one side, and the defense mechanisms (mucosal barrier) on the other. A rise in acid secretion, or weakness of the barrier is likely to start peptic ulceration.

Acute gastric dilatation

This is a condition where the stomach loses its tone and rapidly dilates to reach an enormous size, sometimes filling the abdominal cavity to such an extent that other viscera are totally obscured. The stomach becomes filled with air and fluid which is dark and foul. The source of the fluid is mainly from the intravascular compartment which is depleted and the patient passes into hypovolaemic shock.

The basic pathologic feature, which is loss of tone of the gastric wall, cannot be easily explained but fortunately can often be prevented. This condition can occur postoperatively especially after pelvic operations, splenectomy, and cholecystectomy, but very rarely after gastric surgery. A few cases have been reported during labour and in patients immobilized in plaster casts. It is also seen as a terminal event in patients suffering from debilitating or toxæmic states.

There is a lot of similarity between acute gastric dilatation and paralytic ileus.

1. Both occur postoperatively.
2. Caused by GI atony.
3. Preventable.
4. Vomiting and distension.
5. Treatment is the same, viz, drip and suck. This means IV fluid drip to correct deficits and provide needs, and nasogastric suction.

Clinical features

▪ Early cases

- Hiccough
- Upper abdominal discomfort.
- Tachycardia.

The condition should be diagnosed at this stage and gastric decompression by nasogastric tube instituted.

▪ Neglected cases

- Effortless vomiting of dark foul smelling fluid.
- Picture of hypovolaemic shock.
- Upper abdominal distension.
- Succession splash.
- Aspiration pneumonia may occur.

Prevention

The placement of a nasogastric tube after major abdominal surgery prevents acute gastric dilatation.

Treatment

- Insertion of a nasogastric tube and continuous aspiration.
- Attention to fluid, electrolytes and acid-base balance.

Peptic ulcer disease - general considerations

Nature

A peptic ulcer is caused by acid-pepsin digestion of mucous membranes and only occurs when there is a functioning gastric mucosa capable of secreting both acid and pepsin.

Sites (Fig. 30. 8)

An acid-pepsin secreting mucosa does not digest itself but will do so to adjacent mucous membranes of the following sites

1. Duodenum (commonest)
2. The alkaline gastric antrum
3. The jejunum after a gastrojejunostomy
4. The lower oesophagus
5. Rarely in the small intestines adjacent containing ectopic gastric mucosa.

Acute peptic ulcer

Types

1. Multiple erosions. These occur in the body and fundus of the stomach in patients who are receiving non steroidal anti-inflammatory drugs (NSAID). Being antiprostaglandins these drugs weaken the mucosal barrier.

They are multiple, shallow and punched out. They vary in size from 1 mm (erosions) to 1 cm or

more (Fig. 30.9). They are usually limited to the mucosa and submucosa and after they heal there is no evidence of scarring.

Clinically They present as indigestion, but they are seldom recognized at this stage. It is only when they bleed that the case for an upper endoscopy becomes urgent when they will be recognized.

2. True stress ulcers. These are multiple erosions that if not promptly recognized and treated, coalesce to become the condition known as acute haemorrhagic gastritis that causes severe and intractable upper gastro-intestinal haemorrhage.

Stress ulcers are seen in ICU patients, in those with severe trauma, after major burns (Curling's ulcer), with severe acute pancreatitis and with shock particularly septic shock.

The pathogenesis is uncertain but they represent as do multiple erosions a failure of the gastric mucosal barrier either due to a vascular injury with ischaemia of the mucosa or are due to reflux of bile into the stomach allowing hydrogen ion to back-diffuse to the mucous membrane.

Diagnosis

Diagnosis is by endoscopy.

Treatment

Prophylaxis patients who are at high risk of developing stress ulcers should be receive either proton pump inhibitors or H₂ blockers. Intraluminal antacids and sucralfate may be used.

Treatment

- Discontinuation of the offending drug, if any.
- Correction of the shock state.

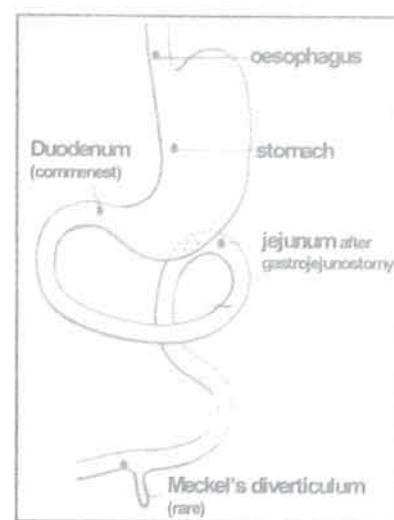


Fig. (30.8) Sites of peptic ulceration

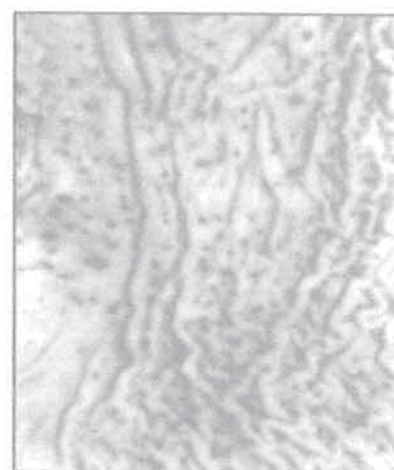


Fig. (30.9) Acute gastric erosions

- Proton pump inhibitors as omeprazole or H_2 blocking agents. These are administered either by the oral or the IV route, according to the severity of the condition.
- Intraluminal antacid to maintain an intraluminal pH of at least 5 may be effective in some cases.
- Mucosal protectors as sucralfate.
- Severe bleeding requires blood transfusion and control of blood loss. The latter can be achieved by endoscopic methods (e.g., adrenaline injection in submucosa or LASER coagulation).
- If all previous measures fail, urgent gastrectomy is the last resort.

Chronic duodenal ulcer

Aetiology

Multiple factors share in the initiation and maintenance of chronic duodenal ulcer, but what is well established is that the disease is associated with increased gastric acidity.

1. ***Helicobacter pylori*** is found in the mucosa of 85% of duodenal and 75% of gastric ulcer patients. *Helicobacter* produces urease enzyme which transforms urea to ammonia, this causes alkalinity of the antrum with increased production of gastrin. Eradication of this bacterium from the stomach markedly reduces the recurrence rate following medical treatment.
2. **Non-steroidal anti-inflammatory drugs (NSAIDs):** They act by decreasing mucosal blood flow due to prostaglandin inhibition. They also prevent healing by inhibiting the proliferation of mucosal cells.
3. **Diseases associated with increased gastrin production**
 - a. Zollinger Ellison syndrome. This is a rare condition where a pancreatic tumour secretes excessive amounts of the hormone gastrin. Less commonly the syndrome is caused by hyperplasia of G cells in the antrum. The high gastrin level causes marked elevation of hydrochloric acid production.
 - b. Hyperparathyroidism This is an unusual cause of ulcers where the high calcium level stimulates excessive gastric production.
4. **Genetic predisposition** this acts by producing a large parietal cell mass, nearly double the normal.
5. **An increased vagal tone**, maximum at night. This is evidenced by the increased amount of overnight gastric acid secretion.

Smoking will most probably not cause an ulcer, but it may delay its healing.

Pathology

Gross appearance

- **Size.** These ulcers are usually small.
- **Site.** They occur in the first part of the duodenum more commonly on the posterior wall and less so on the anterior. Sometimes two ulcers occur one on the anterior the other on the posterior wall, the so called "kissing ulcers" (Fig. 30.10).
- **Induration.** Repeated attempts at healing will result in excessive fibrosis leading to gross deformity of the

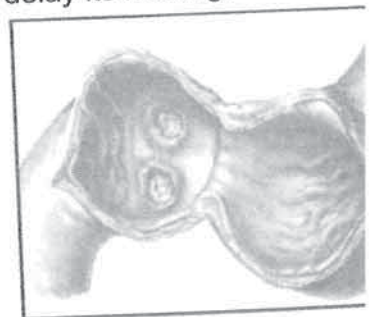


Fig. (30.10). Kissing ulcers

duodenum which is seen at operation and in serial radiographs of the duodenum. An active ulcer is associated with marked induration surrounding it. This is sometimes mistaken for malignant disease by the unwary.

- **Edge and floor.** The ulcer shows sloping edge and a floor covered by a thin layer of granulation tissue.
- **Base.** Its base usually lies in the muscle coat or may even transgress the duodenum to lie in the pancreas.

Complications. Anterior ulcers may perforate while posterior ulcers may bleed because of their close proximity to the gastroduodenal artery or its branches. Dense fibrosis may be a cause of pyloric obstruction (gastric outlet obstruction).

Clinical features

Type of patient

Duodenal ulcers tend to occur predominantly in young active persons. These appear usually well fed and plump with no apparent signs of ill health.

Symptoms

1. **Pain.** This is the leading symptom of duodenal ulcer.
 - a. It is deep seated in the epigastrium and often described as boring.
 - b. It tends to occur a few hours after meals (hunger pain). Undiluted gastric acid will pass to the duodenum, irritates the ulcer and produces pain. This usually occurs at night. This night pain is highly characteristic of duodenal ulceration.
 - c. Duodenal ulcer pain is relieved by food and by antacids. Typically these patients carry with them biscuits that they consume when pain comes on.
 - d. Ulcer pain that is referred to the back usually indicates penetration of the pancreas.
2. Nausea and vomiting are not prominent features of uncomplicated ulcers.
3. **Preiodicity.** Ulcer pain occurs during periods of activity of the ulcer which are of variable length but usually last for a few weeks followed by a period of quiescence which lasts for a few months. During periods of quiescence these patients are quite healthy.
4. A duodenal ulcer sometimes presents by one of its **complications**
 - a. Chronic bleeding, causes anaemia with easy fatigue and pallor.
 - b. Acute bleeding shows by either melaena and/or haematemesis with shock.
 - c. Pyloric stenosis presents with repeated vomiting.
 - d. Perforation of an ulcer will lead to sudden severe pain of peritonitis.

Signs

Localized epigastric tenderness may be present.

Investigations

1. Endoscopy should be carried out in all patients suffering from typical ulcer dyspepsia. By endoscopy the ulcer is directly visualized (Fig. 30.11), an advantage that radiology does not possess.
2. Barium meal used to be the main tool for the diagnosis of duodenal ulcer. It has now been relegated to second place after endoscopy. However, it still is used in some cases where



Fig. (30.11) Duodenal ulcer as shown on endoscopy

there is difficulty in diagnosis or when endoscopy is not readily or expertly available. It is important to obtain serial photographs of the duodenum for the proper diagnosis of ulceration. A fixed deformity of the normally triangular duodenal cap indicates fibrosis. Deformity may be extreme trefoil deformity (Fig. 30.12). The ulcer itself may show when a small speck of barium will remain lodged in the ulcer crater even after the passage of barium. An ulcer may also show as a fixed out-pouching of the duodenal cap (Fig. 30.13). Filling of the duodenum with barium in these cases is difficult for the duodenum is irritable and tends to empty itself very rapidly.

3. Investigations to detect *Helicobacter pylori*. This can be tested by a breath test, by serology, or more commonly by culture from biopsy material.

Treatment

Medical treatment

The treatment of uncomplicated duodenal ulcer is essentially medical. The aim of treatment is to reduce acidity so as to heal the ulcer and to maintain it healed.

- NSAIDs are prohibited.

Medications

- Proton pump inhibitors (PPIs) as oral omeprazole (20 mg/day) and lansoprazole. They markedly reduce gastric acidity in a matter of days. IV omeprazole is used in emergencies. Long-term therapy with PPIs is costly.
- H₂ receptors blockers such as cimetidine, ranitidine and famotidine. These also reduce gastric acid secretion allowing prompt healing of the ulcer. The usual dose of oral ranitidine is 150 mg twice daily.

- Under these regimens the ulcer heals within 6 weeks, but full dose treatment continued for a few weeks more. At this time endoscopy should be repeated to evaluate healing. The main disadvantage of medical treatment for a duodenal ulcer that once the drugs are stopped, there is a high rate of symptoms.

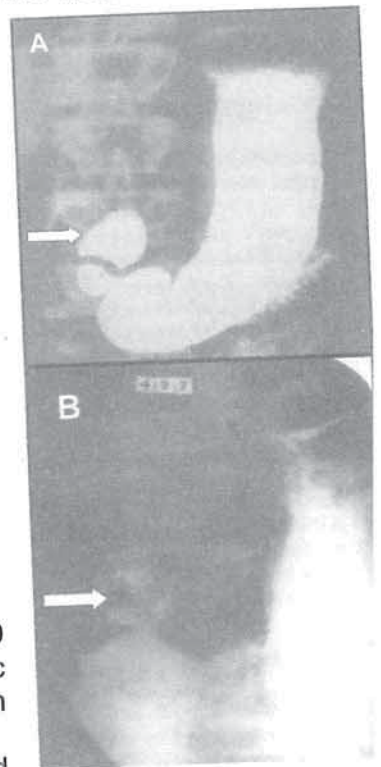


Fig. (30.12) Barium meal shows (A) Normal duodenal cap. (B) Trefoil deformity.

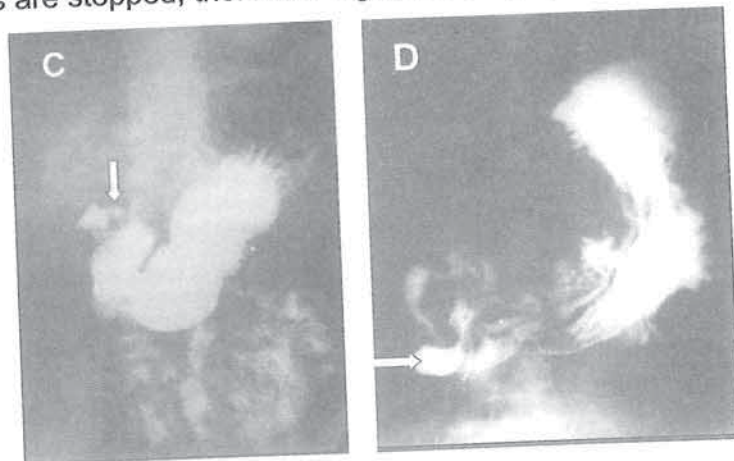


Fig. (30.13) Chronic duodenal ulcer. C. Ulcer crater. D. Fixed outpouching of duodenal cap

Eradication of *Helicobacter pylori*. Eradication of this bacterium significantly reduces the recurrence rate following medical treatment. A triple drug therapy consisting of metronidazole, tetracycline or amoxicillin and bismuth compounds given for 2 weeks gives excellent eradication results in 90% of cases. Testing for *Helicobacter* should be repeated 4 weeks after the cessation of treatment.

Surgical treatment

Indications for surgery

After the development of the aforementioned drugs, the need for elective surgery has much decreased.

1. Failure of medical treatment to control symptoms.
2. Relapses after repeated attempts of adequate medical treatment.
3. Financial inability and non compliance to long-term treatment are relative contraindications.
4. A duodenal ulcer presenting by fibrotic pyloric stenosis requires surgery for its cure.

Aim of surgery for duodenal ulcer is not only to heal the ulcer, but also to provide adequate protection against recurrence.

The principle of surgical operations for a duodenal ulcer is to achieve permanent and adequate reduction of gastric acid secretion by either

1. Abolishing the nervous phase of gastric acid secretion by vagotomy which may be
 - a. Truncal
 - b. Selective.
2. Abolishing the hormonal phase of gastric acid secretion by antrectomy. Some surgeons combine vagotomy and antrectomy.
3. Reduction of the parietal cell mass by a subtotal gastrectomy.

In practice the commonly performed operations nowadays are

1. **Truncal vagotomy and drainage.** The trunks of the vagus nerve are exposed and cut, thus denervating the stomach and abolishing entirely the pathway of gastric secretion (Fig. 30.14). Immediate reduction of gastric HCl secretion occurs in the order of 80% although by the passage of time this decreases to about 50% or even less. However, this does not compromise the late results of surgery. A complete vagotomy is associated with recurrence rate of about 5-10%. Truncal vagotomy has two undesirable side effects; one is motor denervation of the stomach, the other is denervation of the entire abdomen except for the hind gut. The first side effect can be compensated for by performing a drainage procedure in combination with truncal vagotomy. A choice of these procedures exists. There is pyloroplasty, or gastrojejunostomy.

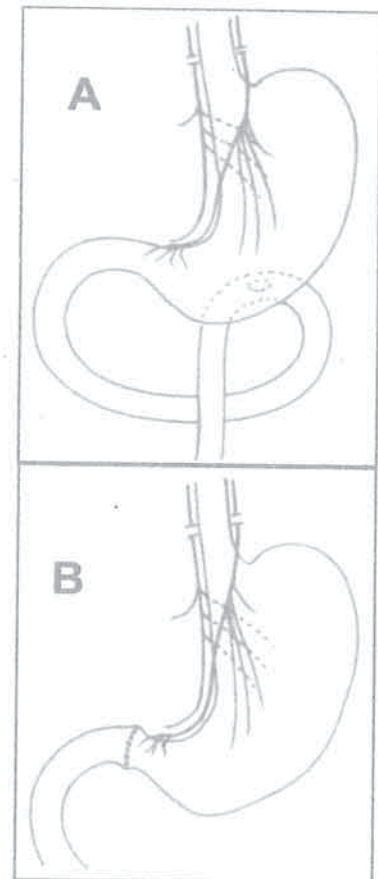


Fig. (30.14) (A) Truncal vagotomy and gastrojejunostomy (B) Truncal vagotomy and pyloroplasty

Gastric drainage procedures

These procedures are an essential addition to truncal vagotomy.

- **Gastrojejunostomy.** Creating a stoma between a dependent part of the stomach and the proximal jejunum (Fig. 30.14A).
 - **Pyloroplasty.** This operation destroys the pyloric sphincter by cutting it. The pylorus is divided longitudinally and sutured transversely (Fig. 30.14.B and 30.15).
2. **Highly selective vagotomy** is designed to denervate only the body and fundus of the stomach, i.e. the acid secreting parts of the stomach leaving the pylorus and its antrum fully innervated, thus preserving gastric emptying and avoiding the necessity of a drainage procedure. The operation consists of ligating all the branches of the left gastric artery to the stomach because the secretory fibres run in close association with them, but stopping short of the branches of the antrum (The crow's foot) Dissection should be kept close to the lesser curvature to avoid injury to the nerves of Latarjet (Fig. 30.16).

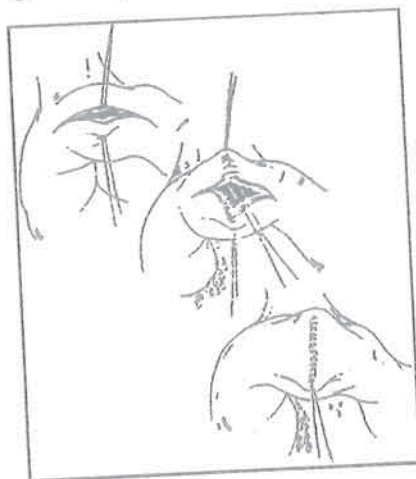


Fig. (30.15) Pyloroplasty

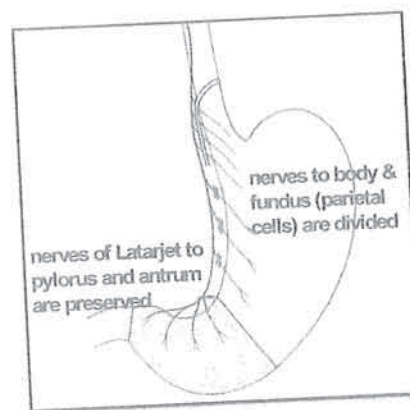


Fig. (30.16) Highly selective vagotomy

Chronic gastric ulcer

Types

There are three types of gastric ulcers (Fig. 30.17)

- Type I ulcers occur in the body of the stomach on the lesser curvature (60%).
 - Type II ulcers occur in association with a duodenal ulcer (20%). The duodenal ulcer develops first and causes the gastric ulcer by inducing duodenal deformity and gastric stasis.
 - Type III ulcers occur in the antrum or pyloric canal (20%).
- Type II and III ulcers are associated with gastric hypersecretion and have the same aetiology as duodenal ulcers (see before).
Type I gastric ulcers are preceded by gastritis that is associated with damage to the gastric mucosal barrier. They are not caused by high acid production, as these patients tend to have lower than normal acid secretion.

Aetiology

The aetiology of this disease is not firmly settled. The causes of gastritis that precede ulceration are:

1. Reflux of bile into the stomach causing damage to the gastric mucosal barrier allowing hydrogen ions to back diffuse and cause mucosal damage.
2. Antral stasis due to defective gastric emptying resulting in hypergastrinaemia.
3. *Helicobacter pylori* was shown to produce gastritis, but its exact aetiological role has not been established.
4. NSAIDs are associated with an increased risk of gastric ulcer particularly if combined with *Helicobacter* infection.
5. Smoking, aspirin and alcohol are associated with gastric ulcers. Smoking in particular inhibits ulcer healing.

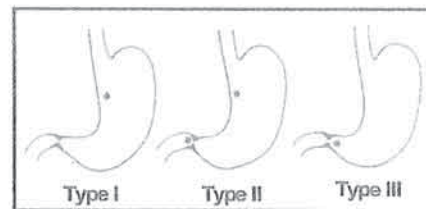


Fig. (30.17) Types of chronic peptic ulcers of the stomach

Incidence

This disease is much less prevalent globally than duodenal ulcer. Males outnumber females, but by a smaller margin than in duodenal ulcer. The disease occurs at an older age than does duodenal ulcer, but exceptions occur.

Pathology

Gross appearance

- **Size.** Gastric ulcers tend to be larger than duodenal ulcers.
- **Site.** They occur at the junction of the body and antrum on the lesser curvature (Fig. 30.18). Some ulcers are saddle-shaped overriding the lesser curvature. An ulcer situated away from the lesser curvature is highly suspicious of malignancy. Rarely, there is a combination of a duodenal and a gastric ulcer. In these cases the duodenal ulcer occurs first then the gastric ulcer follows because of the development of gastric stasis.
- **Edge and floor.** The ulcer shows sloping edges and a floor covered by granulation tissue.
- Its **base** usually lies in the muscle layer but may lie in any tissue the ulcer has penetrated, e.g. the pancreas.
- There is marked induration around it.

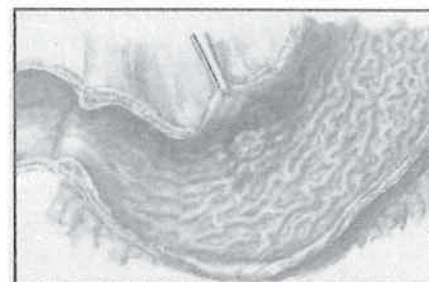


Fig. (30.18) Usual site of a chronic peptic ulcer of stomach

Complications

- Posterior ulcers penetrate the pancreas while high anterior ulcers penetrate the liver. Saddle-shaped ulcers can penetrate both the pancreas and the liver.
- Gastric ulcers may perforate. If perforation occurs posteriorly it occurs in the lesser sac and causes one type of pancreatic pseudocyst.
- Obstruction. Hour-glass stomach.
- **Malignancy and gastric peptic ulcer.** The consensus of opinion nowadays is that these ulcers only very rarely turn malignant, this being in less than 1% of cases. Many of the previous reports indicating a change to malignancy of a benign gastric ulcer were actually reporting on gastric cancer appearing as an ulcer on the lesser curvature.
- The diagnosis of a gastric ulcer is not problematic but the problem is to determine with a high degree of certainty whether that ulcer is malignant or not.

Clinical features (Table 30.1)**Type of patient**

Patients suffering from a gastric ulcer are usually elderly or old and appear thin and underweight.

Symptoms

1. **Pain.** Similar to duodenal ulcer in character and location, it is more intimately related to the intake of food so that the patients clearly perceive the relationship between food and pain and are therefore afraid to eat and consequently lose weight. Pain referred to the back usually signals penetration. Gastric ulcer pain is relieved by antacids and does not occur when the stomach is empty, therefore, night pain as in duodenal ulcer is not a feature. If antacids fail to relieve the pain, the possibility of malignancy or penetration arises.
2. **Nausea and vomiting** are features of this disease. Vomiting brings relief and may be self induced.
3. Although less well marked than in duodenal ulcer, still a gastric ulcer shows periodicity, the loss of which may indicate malignancy.
4. A gastric ulcer sometimes presents with one of its **complications**. Bleeding and perforation occur during periods of activity while fibrous contractures produces hour-glass stomach.

Signs

Physical examination will only reveal localized epigastric tenderness. Tenderness differentiates it from malignancy.

Investigations

1. **Endoscopy** is the principal investigation. The ulcer is directly visualized and its characteristics noted. Multiple biopsies (at least four) should be obtained from the ulcer to exclude malignancy (Fig. 30.19 and 30.20).
2. **Barium meal** has receded to second place after endoscopy in the investigation of these cases. An ulcer will appear as an outpouching (niche) on the lesser curvature or as a crater which remains filled with barium after its passage from the stomach. The niche of a benign ulcer projects outside the line of the stomach (Figs. 30.21). An area of spastic muscle on the greater curvature opposite the ulcer is frequently seen (ulcer notch). There is tenderness over the site of the ulcer. It is important to obtain a mucosal relief pattern before filling the stomach with barium. In this

Table. 30.1. Clinical differences between a chronic duodenal ulcer (DU) and a chronic gastric ulcer (GU).

	DU	GU
Age	3rd & 4th decades	Past middle age
M:F	5:1	2:1
Pain	2-3 h. after meals. Nocturnal and hunger pain	Shortly after meals
Nausea & vomit.	May occur	Present Brings bile
Periodicity	Marked	Less marked
Appetite	Good	Afraid to eat
Weight	May be overweight	Weight loss



Fig. (30.19) All chronic gastric ulcers should be biopsied to exclude malignancy



Fig. (30.20) Gastric ulcer shown on endoscopy

way the sign of rugal convergence will be seen.

3. Tests for *Helicobacter pylori*.

Treatment

Medical treatment

This is almost exactly similar to duodenal ulcer but because of the risk of being malignant, one must repeat that it is essential to exclude malignancy before embarking upon medical treatment.

Repeated endoscopy should be carried out after 6 weeks regardless of symptomatic improvement. If the ulcer shows no attempt at healing, it should be considered malignant and surgery is performed. If partial healing is seen, it would then seem reasonable to persist for another 2 months (never more than 6 months) with medical treatment and repeat endoscopy. It should be noticed that some patients with gastric cancer may improve on medical treatment.

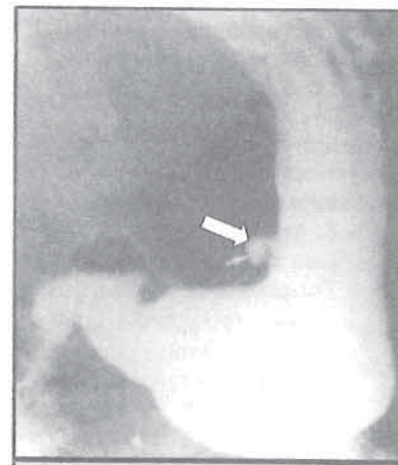


Fig. (30.21) Barium meal shows an ulcer notch on lesser curve

Surgical treatment

Indications for surgery

1. Failure of healing to start at 6 weeks or of complete healing at 6 months after medical treatment.
2. Uncertainty about the absence of malignancy.
3. Bad patient compliance and financial factors.
4. Complications of gastric ulcer may require surgery.
 - a. Fibrous contractures.
 - b. Perforation.
 - c. The occurrence of bleeding while the patient is under medical treatment.

Aim of surgery for benign gastric ulcers

The aim of surgery is to remove the ulcer or the entire ulcer bearing area and to ensure proper gastric emptying.

Operation

Partial gastrectomy is the standard procedure. It removes the ulcer together with the entire ulcer-bearing area. The operation is ended by gastroduodenal anastomosis, Billroth I (Fig. 30.22).

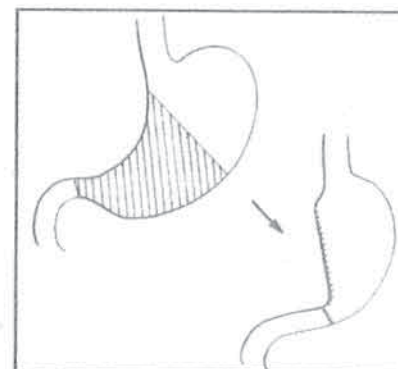


Fig. 30.22. Partial gastrectomy and Billroth I anastomosis. The ulcer-bearing area on lesser curve is removed

Complications of peptic ulcer

- **Acute** complications that occur during periods of ulcer activity.
 - Bleeding.
 - Perforation.
- **Chronic** complications that occur insidiously.
 - Fibrous contractures of ulcers produce pyloric stenosis, and hourglass stomach.
 - Penetration.
 - Malignant transformation of a gastric ulcer.

Perforation

Pathology

Depending on the degree of aggressiveness of the ulcer, perforation will either occur very rapidly or more slowly. Rapid perforation will result in generalized peritonitis. Slower perforations are small and are rapidly sealed.

Perforation usually occurs in anteriorly situated ulcers. Rarely a posterior gastric ulcer perforates into the lesser sac.

At the time of perforation duodenal or gastric contents burst into the peritoneal cavity and set up an inflammatory response; chemical peritonitis, the contents being sterile. In a matter of a few hours swallowed bacteria (with saliva) and bacteria from the gut seeping through the perforation and gaining access to the peritoneal cavity will turn chemical peritonitis into a full fledged bacterial peritonitis.

The inflammatory exudate at first collects in the supracolic compartment of the peritoneal cavity, but soon spills to the infracolic compartment by way of the right paracolic gutter (the left being closed by the phreno-colic ligament) and may remain localized to the right iliac fossa for some time giving conflicting clinical signs and sometimes wrongly diagnosed as acute appendicitis (Fig. 30.23).

Small perforations may be sealed rapidly without much soiling of the peritoneal cavity.

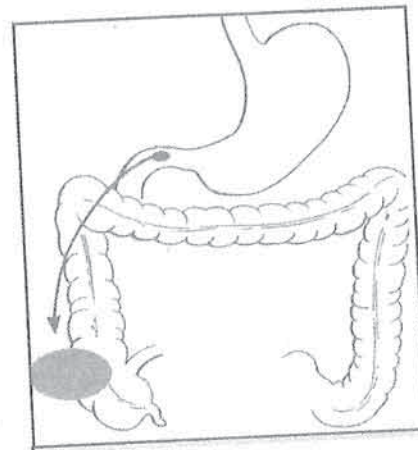


Fig. (30.23); Perforated duodenal ulcer may simulate appendicitis

Clinical features

These are usually described in 3 stages.

1. **Stage of chemical peritonitis**
 - At the time of perforation the patient experiences sudden severe pain starting in the epigastrium, and later becomes generalized.
 - The patient often collapses especially with a large perforation.
 - There is tachycardia, but the temperature is often subnormal.
 - Abdominal examination reveals board-like rigidity, and tenderness mainly in the epigastric region. Sometimes there is tenderness in the right iliac fossa which may be misdiagnosed as acute appendicitis. Percussion reveals attenuated liver dullness and auscultation reveals a silent abdomen.
2. **Stage of illusion** The peritoneum reacts to chemical peritonitis by secreting copious amounts of fluid, and thus lessening the chemical irritation. The patient feels better with less pain and less rigidity.
3. **Stage of bacterial peritonitis** Secondary infection of the peritoneal cavity leads to septic peritonitis. Pain increases and there is rising temperature and tachycardia. Abdominal examination reveals generalized tenderness, rigidity and progressive abdominal distension.

Differential diagnosis

1. From causes of acute upper abdominal pain.
 - Acute cholecystitis.
 - Acute pancreatitis.
 - Mesenteric vascular occlusion.
 - Leaking aortic aneurysm.

- Basal myocardial infarction.
- 2. From acute appendicitis if it presents by right iliac fossa pain.

Investigations (should be urgent)

1. **Chest x-ray** done in the erect position usually reveals air under the cupola of the diaphragm (Fig. 30.24).
2. **Abdominal ultrasound** will reveal peritoneal fluid.
3. If perforation is suspected and plain x-ray failed to reveal gas under the diaphragm, a gastrograffin meal can be performed.

Treatment

Treatment is by urgent operation after correction of hypovolaemia, if present, and of fluid and electrolyte imbalance. Preoperative preparation includes

1. A Ryle's tube is passed and continuous aspiration instituted.
2. IV fluids are set guided by urine output and by electrolyte and pH estimations.
3. IV antibiotics are given.
4. IV H₂ receptor blocking agents or omeprazole are given.
5. Careful attention to respiration which is often compromised.

At operation the simplest procedure is probably the best choice. Closure of the perforation with an omental patch is simple and adequate (Fig. 30.25). Thorough peritoneal toilet must be conducted and the abdomen is closed with drainage. In the case of a perforated gastric ulcer a biopsy must be obtained from it.

After treatment Patients are treated medically for their ulcer disease.

Subacute perforation. This is a small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed. It gives the same early signs but much reduced. If diagnosed correctly it can be treated conservatively. Chronic perforation means penetration into an adjacent structure as the pancreas. It is suspected when the pain becomes more or less persistent and referred to the back.

Bleeding

Pathogenesis

Bleeding from a peptic ulcer always occurs but only on a microscopical level and is considered part of the disease process. Bleeding as a complication, haematemesis and melaena occurs due to the ulcer meeting and eroding a large vessel at its base. This at first results in a small erosion of the vessel which bleeds a little before being sealed off. This may be repeated until a large rent in the vessel wall results in massive bleeding. This results clinically in the patient having a small dark brown-coffee ground-vomit which may be repeated until massive haematemesis occurs.

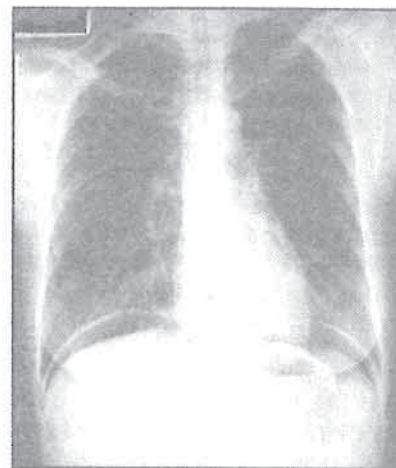


Fig. (30.24); Chest X-ray, in erect position, shows air under diaphragm.



Fig. (30.25) Closure of a duodenal ulcer perforation by an omental patch

If the ulcer bleeds as melaena, the small premonitory vomits do not occur. The patient feels faint and may collapse, then he passes loose black tarry stools. If bleeding was massive, red blood may be passed with stools.

Clinical features

Haematemesis and melaena.

The aim is to evaluate the amount of blood loss and its rapid replacement and at the same time to determine the source of bleeding in order to start attempts to stop it.

Small bleeds cause no alteration in the patient's general condition except for apprehension but they require hospitalization and should receive exactly the same diagnostic and treatment policies as large bleeds. More severe haemorrhage will cause the patient to become pale, cold, faint with a rapid thready pulse and low or dropping blood pressure (Chapter 4).

Haemoglobin and haematocrit estimations become useful only after a few hours when haemodilution has already occurred. The previous signs call for extremely urgent replacement by blood or a plasma expander, opiates are given to allay anxiety.

Conservative treatment

Most of these cases stop bleeding spontaneously due to clot formation in the base of the ulcer obstructing the rent in the artery and therefore conservative treatment should always be considered whenever possible.

- Admission to hospital and better to an intensive care unit.
- High-flow oxygen by mask.
- Correction of hypovolaemia by IV crystalloids and blood transfusion.
- Careful monitoring of urine output, pulse, blood pressure and haematocrit.
- Nasogastric tube.
- IV PPIs.

Once the general condition of the patient is stabilized, urgent endoscopy is performed. The ulcer is usually covered by a blood clot or there may be an actual spurting artery. Gastroscopy provides a diagnosis of the cause of bleeding and can also serve achieve haemostasis.

Indications for endoscopic haemostasis

- The ulcer is seen to be actively bleeding.
- Stigmata of recent bleeding because they indicate likelihood for rebleeding.
- A visible vessel in the ulcer base (Fig. 30.26).
- Fresh clot covering the ulcer.

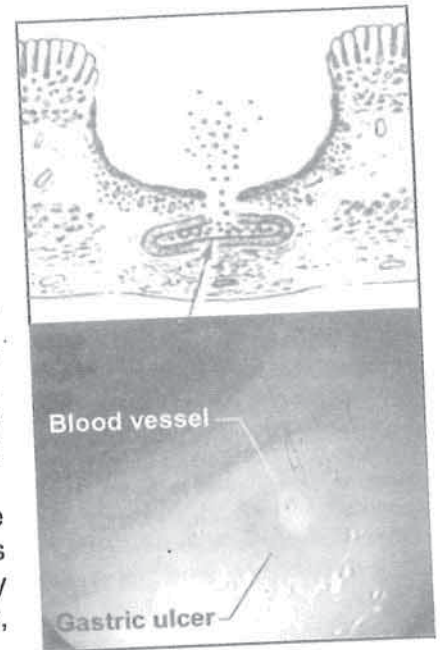


Fig. (30.26) Endoscopic view shows a gastric ulcer with a visible vessel in its base. This is an indication for endoscopic haemostasis

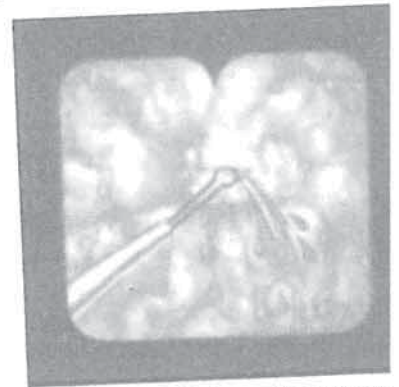


Fig. (30.27) Endoscopic diathermy coagulation of a bleeding ulcer

Methods of endoscopic haemostasis. Fine instruments are introduced through the channel of the gastroscope, reach the ulcer and arrest bleeding by either laser coagulation, thermal coagulation, diathermy coagulation (Fig. 30.27), or by injection of the ulcer base by adrenaline or alcohol.

The following cases usually respond favourably to conservative treatment

1. Young patients under 45 years.
2. Minor bleeds.
3. Short history of dyspepsia.
4. Recent history of ulcerogenic drug intake, for withdrawal of the drug may be all that is required.

Surgical treatment

The decision to operate should be taken early (within 48-72 hours) for the results of late surgery in these cases are poor with a high mortality. The patient should have more than one IV line open and running. A nasogastric tube is passed and the stomach is washed and emptied.

Indications

Absolute indications

1. If a patient under adequate medical treatment bleeds, then conservative treatment has nothing more to offer and operation becomes mandatory.
2. If the initial bleed was severe, 2000 ml or more.
3. If bleeding continues as evidenced by the need to transfuse large volumes (1000 ml) of blood per day to maintain stability.
4. If bleeding recurs while the patient is still in hospital.

Relative indications

1. Old arteriosclerotic patients should be considered for surgery because they cannot withstand the ill effects of shock but withstand the lesser brunt of surgery better. Their arteries are also incapable of retraction enough to stop haemorrhage.
2. Those with a long history of ulcer disease do not respond well to conservative treatment.
3. Those with associated serious disease. It must be remembered that the risk of operation in these patients is less than the risk of shock.

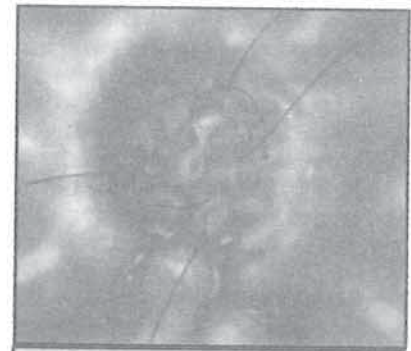


Fig. (30.28) Under-running sutures to stop bleeding

Operation

- For a duodenal ulcer, the pylorus is opened longitudinally to expose the bleeding ulcer. Haemostasis is achieved by under-running sutures (Fig. 30.28). The pylorus is then closed transversely thus performing a pyloroplasty. A truncal vagotomy is added to reduce acid secretion.
- For a bleeding gastric ulcer gastrectomy is the standard operation.

Pyloric stenosis

Pyloric stenosis is caused by excessive fibrosis around a duodenal or a juxtapyloric ulcer resulting in gastric outlet obstruction. This usually occurs over a period of years of ulcer dyspepsia, although some cases occur silently.

Clinical features

1. Vomiting. The patient vomits copious amounts of foul dark fluid characteristically in the evening. The patient recognizes undigested food eaten the previous day or even earlier. Vomiting is projectile and does not usually contain bile.
2. The patients are usually capable of eating breakfast then have a small lunch but are unable to eat in the evening because of a sense of discomfort and fullness which is due to a full stomach. The initial periodic ulcer pain is lost and is replaced by epigastric discomfort.
3. On examination, these patients are usually under weight, ill nourished and may be dehydrated. Abdominal examination may show the contour of the enlarged stomach with visible peristalsis passing from left to right. A succussion splash can often be heard.
4. Patients with longstanding pyloric obstruction will usually have serious electrolyte disturbances including hyponatraemia, hypokalaemia, alkalosis and hypocalcaemia which may lead to tetany.

Differential diagnosis

- Carcinoma of the pyloric antrum.

Investigations

To diagnose that the pylorus is obstructed is not a great challenge, for the clinical picture is clear, but investigations are aimed at the cause of obstruction and at assessment of the general status of the patient.

1. Haemoglobin, haematocrit and serum electrolytes estimations are performed. Bicarbonate estimation is also done.
2. Endoscopy will show a stenosed pyloric ring which will be inactive and will not pass the tip of the endoscope. The main value, however, is to detect the presence of a tumour at the pyloric end of the stomach and to obtain biopsies from it if found. A point to remember is that endoscopy can only be done on an empty stomach and therefore should follow a period of fasting and of gastric suction-lavage.
3. Barium meal will show the dilated stomach (Fig. 30.29) often reaching the pelvis (soup dish appearance), will show food residue, v reveal delayed gastric emptying, but will often miss a small pyloric carcinoma. Gastroscopy is, therefore essential for diagnosis.

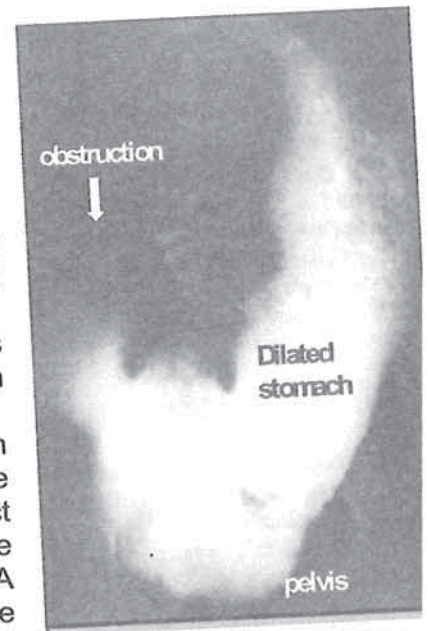


Fig. (30.29) Barium meal shows pyloric stenosis. The stomach is hugely dilated down to the pelvis.

Management

Pyloric stenosis requires surgery, but is not an emergency. Preoperative preparation should be performed. All initial efforts are directed at building up the nutritive state of the patient in order to reduce morbidity and mortality following surgery.

1. The stomach should be sucked and washed through a nasogastric tube.
2. A high protein fluid diet should be given and is supplemented by intravenous alimentation.
3. Correction of fluid, electrolytes (Na, K, Cl and Ca) and acid-base balance (alkalosis) by proper intravenous solutions.
4. Transfusion of packed cells to correct anaemia which is often present.

Some cases of pyloric stenosis occur due to muscle spasm and a marked inflammatory reaction around an active duodenal ulcer. Pain in these cases is prominent. This is usually seen in the young. These cases respond well to medical treatment as for uncomplicated ulcers.

Operation

Truncal vagotomy and gastrojejunostomy is the standard treatment for the condition.

In the elderly or unfit patients, it may be justifiable to perform a gastrojejunostomy alone.

Hour glass stomach

This is due to a cicatrized saddle shaped ulcer on the lesser curvature. It presents by symptoms of gastric obstruction but there will be early satiety and loss of weight. Since it usually occurs insidiously in the elderly, its clinical differentiation is from carcinoma of the stomach. Endoscopy and a barium meal will settle the diagnosis (Fig. 30.30).

Treatment is by partial gastrectomy which removes the distal pouch.

Complications of gastric operations

These operations can be followed by any of the complications known to occur after major surgery, but they also have specific complications of their own.

Early complications

1. **Bleeding** from the anastomotic line is revealed by fresh blood being aspirated through the Ryle's tube. It usually responds to conservative management but if it persists will require re-operation.
2. **Stomal obstruction.** Most cases are due to oedema at the stoma and will respond to aspiration and IV therapy. In some cases a mechanical factor is the cause, e.g. a technical error or obstruction of a gastroduodenal anastomosis by hypertrophied antral mucous membrane or a retrograde jejuno gastric intussusception. Apart from the latter which may reduce spontaneously or after a barium meal, these cases require surgery to correct them. A troublesome complication is seen sometimes after vagotomy in which the stoma is actually patent but the stomach is atonic and does not empty. These cases may require prolonged conservative management by suction and IV fluids.
3. **Duodenal blow-out.** This follows a Billroth II anastomosis (Fig. 30.31). It usually occurs around the fourth day but may occur later. These cases are due to
 - a. A bad choice of operation where a suture line was placed in an inflamed or a densely scarred duodenum.

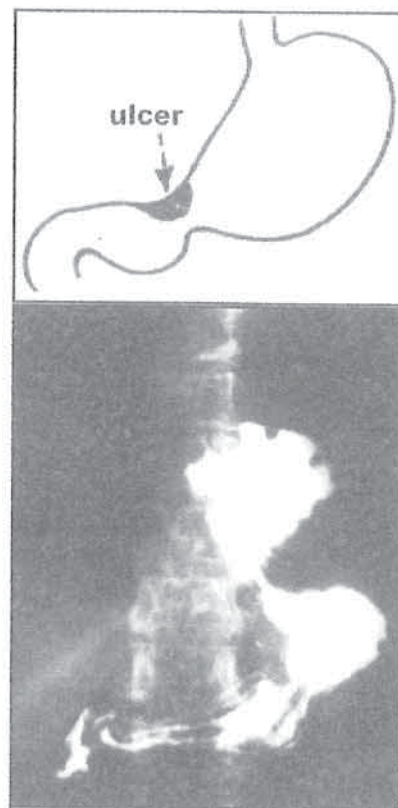


Fig. (30.30) Hour glass stomach

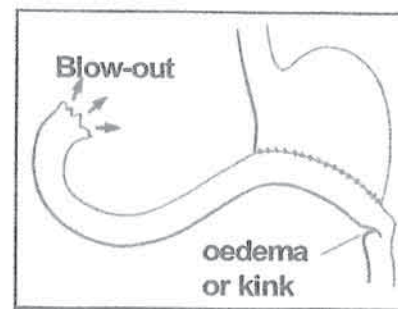


Fig. (30.31) Duodenal blow-out

- b. Technical error. Obstruction of the afferent loop or excessive turning in of the duodenum or a too aggressive dissection resulting in ischaemia. If drainage was provided, duodenal contents will soak the dressings. If no drainage was provided, then acute severe upper abdominal and lower chest pain with apprehension and tachycardia, soon followed by generalized peritonitis will occur. In any case drainage must be provided together with intravenous alimentation. This will cure all cases when there is no distal obstruction. In others reoperation to relieve afferent loop obstruction is required.

4. Post-operative pancreatitis.

Late complications

1. **Recurrent ulceration** It is usually due to inadequate surgery. Rarely it may be due to other causes such as a gastrinoma causing the Zollinger-Ellison syndrome. Missing a vagal trunk, usually the posterior, is the usual cause following truncal vagotomy or missing of the so called "criminal nerve" which is a branch of the vagus nerve running over the lower oesophagus, during the performance of a highly selective vagotomy. Following gastrectomy the usual cause is leaving part of the gastric antrum with the duodenum. This results in a persistent hypergastrinaemia. A recurrent ulcer may be stomal, i.e. on the anastomotic line, or on one side of it whether gastric or jejunal or may be a true recurrence of the original ulcer. Clinical diagnosis is not difficult because of the recurrence of typical ulcer symptoms. Endoscopy is essential for the diagnosis. H_2 receptor blockers or omeprazole will control most if not all of these patients, but recurrence is common after cessation of the drug, so that reoperation is popular. Following vagotomy, looking for and dividing a missed trunk can be tried, but if this is not found, an antrectomy is performed. Following gastrectomy vagotomy is the procedure of choice.

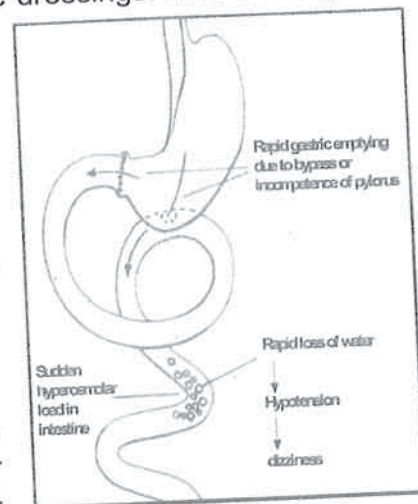


Fig. (30.32) Early dumping

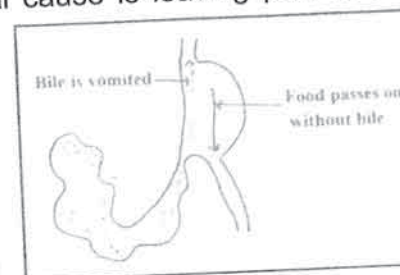


Fig. (30.33) Afferent loop syndrome.

2. **Dumping.** It is a syndrome with two components; vasomotor and gastrointestinal symptoms following meals. If symptoms occur within the first 1/2 hour after meals is known as early dumping or if delayed for about 2-3 hours it is called late dumping. In early dumping (Fig. 30.32), the patient complains of epigastric fullness and pain with nausea and ending in explosive diarrhoea. The vasomotor component is a sense of weakness, flushing and palpitation. This syndrome results from rapid gastric emptying with the delivery of a hyperosmolar solution to the proximal small gut with the result of shift of fluid from the circulatory plasma to the lumen of the proximal small gut resulting in the vasomotor effects. Another responsible factor is the liberation of hormones as serotonin and bradykinins from the distended small intestine and which may also account for the explosive diarrhoea. Most cases are controlled by assuming the supine position. Prevention, however, is important and includes small frequent feeds rich in proteins and fats and less in carbohydrates with liquids only allowed between meals.

Late dumping. The symptoms of late dumping which occur about 2-3 hours after meals are those of hypoglycaemia; sweating, palpitation, tremors, and confusion and which are relieved by carbohydrate ingestion. Hypoglycaemia is brought about by an overshoot of insulin which is caused by rapid delivery of large amounts of carbohydrates to the small intestine. Prevention can be achieved by giving frequent small feeds of a carbohydrate low diet.

3. **Biliary gastritis (alkaline reflux gastritis).** Reflux of bile through a stoma into the stomach causes a diffuse gastritis. This mimics recurrent ulceration with the added symptom of bilious vomiting. Careful endoscopic diagnosis is mandatory for these patients who can only be cured by surgery. Fashioning of a Roux-en-Y anastomosis gives good results.
4. **Afferent loop syndrome** This is due to a technical error resulting in obstruction of the too long afferent loop by twisting (Fig. 30.33). The patient complains of fullness and epigastric pain following meals. This is followed by projectile bilious vomiting which contains no food. Food has stimulated pancreatic, and bile secretion which distend the afferent loop. Pressure increases until obstruction is overcome and the loop empties itself into the stomach. Food having already passed from the stoma is not present in the vomitus. Operative treatment is essential and consists of conversion of the anastomosis to a Roux-en-Y loop.
5. **Postvagotomy diarrhea** A tendency to loose stools follows truncal vagotomy in most cases. Rarely in about 3% of cases, the diarrhoea is watery and explosive. The cause is obscure but may be related to bile acids being delivered too rapidly to the colon due to dysfunction of the ileo-caecal valve. The condition gets better as time goes on.
6. **Gastrojejuno-colic fistula** This is due to a recurrent ulcer following an operation in which a gastrojejunostomy was used. The ulcer will adhere and then penetrate into the colon resulting in a fistula between the stomach, the jejunum and the colon (Fig. 30.34). The symptoms of recurrent ulceration will be modified by the occurrence of severe diarrhoea, ill health, loss of weight, and anaemia. The diarrhoea is due to an enteritis caused by the entry of colonic contents into the jejunum. For its visualization a barium enema (and not meal) is needed. The treatment is surgical. Proximal diverting colostomy is performed at first, followed later by correction of the fistula.
7. **Postgastrectomy nutritional disturbances.** Weight loss, anaemia (iron deficiency or megaloblastic), steatorrhoea, vitamin B deficiency, calcium deficiency, early satiety, and an increased risk of pulmonary TB.
8. Increased risk of stomach cancer following gastrectomy.

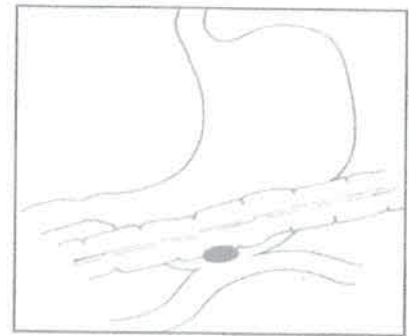


Fig. (30.34) Gastro-jejuno-colic fistula

The changing role of surgery for peptic ulcer

- With better understanding of gastric secretory physiology, powerful gastric acid inhibitors were developed, starting from cimetidine, through the other H_2 receptor blockers to the proton pump inhibitor omeprazole.
- These control peptic ulcer disease very well, debate only exists as regards their long term use. The role of surgery has declined to such a degree that it is now rarely indicated for the control of uncomplicated ulcer disease
- Now the surgeon finds himself dealing with the complications of, rather than, the ulcer itself. The surgeon now usually deals with bleeding, perforation and fibrous contractures.
- Another change has been brought about by the introduction of laparoscopic surgery. Vagotomy has been successfully performed using the laparoscope.

Neoplasms of the stomach

Benign gastric neoplasms

All types of benign neoplasms expected to arise from mucous membrane, muscle, and interstitial tissue occur in the stomach. The commonest is the leiomyoma. It can give vague symptoms and appears as a smooth filling defect in a barium meal (Fig. 30.35). It can bleed or may perforate the stomach. It occasionally gives rise to an epigastric mass. Malignant transformation can occur.



Malignant gastric neoplasms

1. **Adenocarcinoma** is the commonest.
2. **Non-Hodgkin's Lymphoma** and **sarcoma** occasionally occur in the stomach and carry a better prognosis than does carcinoma.
3. **Gastrointestinal stromal tumour.**

Carcinoma of the stomach

Except in Japan and despite the great advances that have occurred in surgery, radiotherapy, and chemotherapy, the prognosis of gastric cancer has remained very bad. The overall 5 years survival rate is about 5%.

Fig. 30.35. Gastric leiomyoma shows as a smooth filling defect on barium meal radiography

Incidence

The incidence of gastric cancer has been falling steadily throughout the world except in Japan. There is no proper explanation for the above mentioned facts.

Aetiology

1. Several factors, mostly dietary, have been implicated, but in general any factor that causes gastritis can cause carcinoma. Examples are tobacco, alcohol, spices, and increased salt intake.
2. Factors that cause achlorhydria such as pernicious anaemia can cause cancer. The presence of nitrates in food leads to the production of N-nitrosamines by the action of bacteria in the achlorhydric stomach. N-nitroso compounds are carcinogenic in animals.
3. The risk of gastric cancer in patients who have chronic H. pylori infection is increased about 3 times.
4. A genetic predisposition. Persons with blood group A are more susceptible.
5. Benign gastric ulcers very rarely turn malignant (see before).
6. Benign gastric neoplasms as gastric polyps.
7. Following gastrectomy. Twenty years after partial or subtotal gastrectomy, the remaining gastric stump is more susceptible to the development of carcinoma. This is due to biliary reflux gastritis.

Pathology

Most tumours (60%) occur in the pyloric antrum (Fig. 30.36) and least of all is the type that affects the stomach diffusely. Recently there is an increase in the incidence of carcinoma affecting the upper third of the stomach and cardia.

Macroscopic types

Pathologists now classify gastric cancer into two types.

- A. **Early gastric cancer**; where only the mucosa or submucosa is infiltrated (Fig. 30.37). This type is only diagnosed if screening programs by endoscopy are performed. Early gastric cancer may be protruding, superficial, or excavating (penetrating).
- B. **Advanced gastric cancer**. This is the usually diagnosed type in clinical practice. It may take the form of
 - a. A fungating cauliflower-like mass (Figs. 30.38).
 - b. An ulcer with raised indurated edges and usually surrounded by smaller ulcers.
 - c. Colloid carcinoma. All layers of the stomach are infiltrated by areolar tissue containing transparent gelatinous substance.
 - d. The diffusely infiltrating variety. "Linitis plastica" in which the wall of the stomach is greatly thickened and indurated while the lumen is greatly reduced (Fig. 30.39). This may occur only in the antrum or may affect the stomach more diffusely. The mucous membrane is intact and the lesion may be missed by endoscopy.



Fig. (30.36) Distribution of stomach cancer

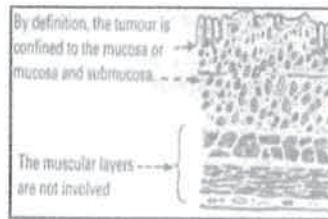


Fig. (30.37) Early gastric cancer

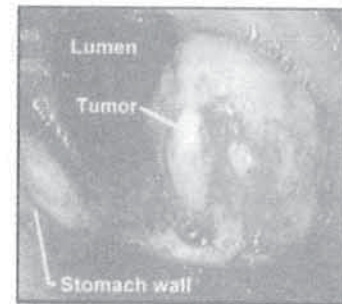


Fig. (30.38) A fungating gastric cancer as shown on endoscopy

Microscopically stomach cancer is an adenocarcinoma with various grades of differentiation, the less the differentiation the worse the prognosis. A colloid carcinoma carries a bad prognosis so does linitis plastica in which it is difficult to find malignant cells under the microscope.

Spread

1. Direct spread is infiltration of the wall of the stomach in depth, in circumference, and in length. The last is important because the spread occurs for some length beyond the edge of the tumour, a point to remember when planning a radical operation. After the gastric wall has been transgressed carcinoma will infiltrate any neighbouring structure as the liver, spleen, pancreas or colon.
2. Lymphatic spread occurs both by permeation and by embolization. It first occurs to perigastric lymph nodes then to more centrally placed groups. A sound knowledge of the lymphatic drainage of the stomach is important in planning radical surgery.
3. Blood stream spread occurs mainly to the liver and very rarely beyond it to bones.
4. Transcoelomic spread occurs to the ovaries in younger females when the ovaries are not yet atrophic. These ovarian secondaries are known as Krukenberg's tumours. Metastases to the Douglas Pouch lead to what is known as Blumer's shelf.

Clinical features

This disease is usually diagnosed at a late stage because of the vague and often mild symptoms it gives leading to late presentation by the patient and more disastrously, to late recognition by the clinician. It is important to stress that a high index of suspicion is needed for its detection at an earlier stage.

For simplification it can be stated that patients with carcinoma of the stomach may fall into one or more of 5 groups.

1. **Dyspepsia group.** Dyspepsia above 40 A person above 40 years who starts to complain of dyspepsia should be fully investigated for the possibility of stomach cancer. The patient has anorexia and has a vague sense of discomfort after meals. Epigastric pain may occur and in late cases may be severe. Nausea may be in evidence and early satiety is common.
2. **Insidious group (Anorexia, Asthenia, Anaemia).** Listlessness, easy fatigue and unexplained weight loss may be pronounced and the patient is found to be anaemic. Unfortunately a large number of these patients will disregard these vague symptoms and will try tonics and digestives while their disease inexorably progresses to an advanced stage.
3. **Mass group.** An epigastric mass. About 30% of patients presenting in this way will be found to harbour an inoperable carcinoma on exploration.
4. **Obstructive group.** Carcinoma occurring at one end of the stomach causes obstructive symptoms and will therefore usually present earlier than the more common variety occurring in the antrum or body. At the cardia it will lead to dysphagia while at the pylorus it causes vomiting (see pyloric stenosis).
5. **Metastatic group.** A hard irregular liver due to secondaries, jaundice, malignant ascites, or an enlarged left supraclavicular lymph node "Troisier's sign", all of which are signs of inoperability.

Haematemesis and melaena are uncommon presentations while perforation is still rare. A patient known to have a gastric peptic ulcer who becomes refractory to treatment should be viewed with suspicion.



Fig. 30.39. Barium meal shows an irregular filling defect of gastric cancer

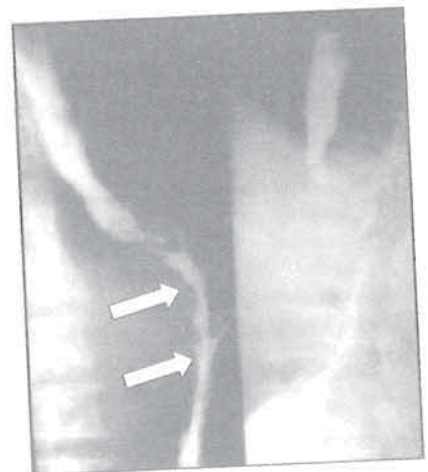


Fig. 30.40. Stomach is narrowed in Linitis plastica as shown on barium meal examination

Investigations

1. Blood picture may reveal micro or macrocytic anaemia.
2. **Barium meal.** Its overall accuracy is about 75%. It is the most accurate for pyloric growths. It is important to obtain a mucosal relief pattern before filling the stomach with barium. Small growth may remain undetected if this is omitted. A barium study should always include views taken in the Trendlenburg's position to detect growths situated in the fundus and cardia. If the lesion is of the cauliflower-like variety, it will appear as a persistent irregular filling defect (Fig. 30.39). If it is a malignant ulcer it will appear as an ulcer niche outside the ulcer bearing area. If the lesion is linitis plastica there will be marked narrowing of the lumen of the stomach but the flow of barium is not interrupted (Fig. 30.40).
3. **Endoscopy** has the considerable advantage of directly viewing the tumour from which multiple biopsies should be taken (Fig. 30.41). Endoscopy can detect tumours at an earlier stage than can radiology.
4. An abdominal sonogram is essential for the detection of liver secondaries. Its role in assessing lymph node involvement is less important.
5. CT scan is more accurate for the detection of lymph node involvement than a sonogram and may be helpful in pre-operative staging of the disease.
6. Estimation of CEA antigen has a better value for prognosis rather than diagnosis.
7. Endoscopic ultrasound can detect the depth of invasion of the gastric wall.
8. Diagnostic laparoscopy.
9. PET-CT scan to detect metastases.

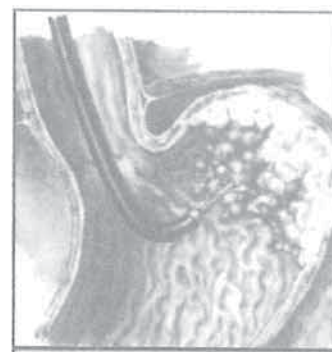


Fig. (30.41) Endoscopic biopsy of gastric cancer

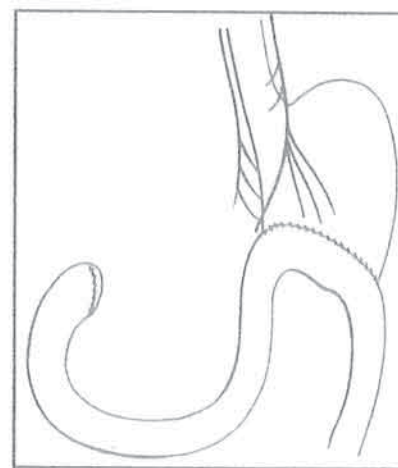


Fig. 30.42. Billroth II (Polya) gastrectomy

Treatment

Early cases. The aim of treatment is cure. For this to be achieved a radical operation has to be performed. This entails removal of the tumour with an adequate safety margin above it of at least 5 cm and 1.5 cm of the duodenum below it, together with both omenta. All the draining lymph nodes must be removed. According to the site of the neoplasm, the extent of resection will be determined:

- (a) For tumours of the lower third of the stomach, a lower radical partial gastrectomy is performed with anastomosis of the remaining upper stomach to the jejunum. This anastomosis is called Billroth II or Polya gastrectomy (Fig. 30.42).
- (b) For tumours of the middle third of the stomach a

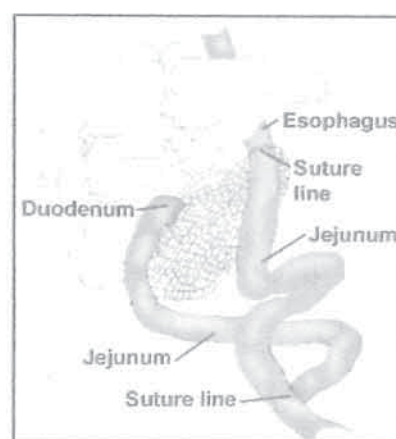


Fig. 30.43. Total gastrectomy and Roux-en-Y oesophago-jejunostomy

total radical gastrectomy is performed and the oesophagus is anastomosed to a Roux-en-Y loop of jejunum (Fig. 30.43).

- (c) Tumours of the upper third are treated by oesophagogastrectomy through a thoraco-abdominal incision to guarantee an upper safety margin. The oesophagus is anastomosed to a Roux-en-Y loop of jejunum.

Patients with incurable or irresectable lesions may have some benefit from various palliative procedures

1. Palliative gastrectomy. If it can be performed, gastrectomy offers the best chance of palliation.
2. For irremovable tumours in the pyloric region an anterior gastrojejunostomy is performed.
3. For irresectable lesions in the upper stomach, an oesophagojejunostomy can be performed.
4. Radiotherapy and chemotherapy. Unfortunately these modalities of treatment offer too little for these miserable patients.

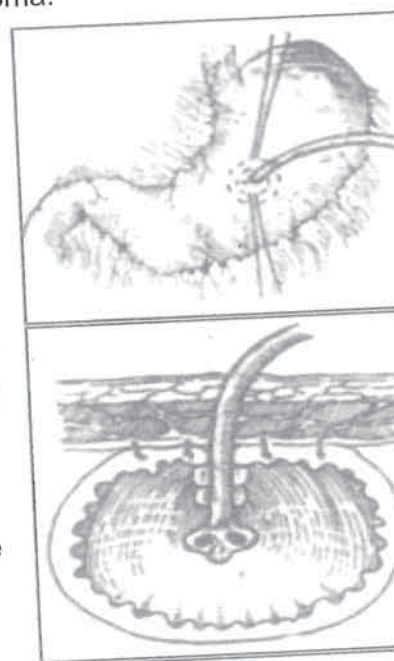
Prognosis

This depends on many factors the most important are the histological type and differentiation, the depth of penetration of the wall and the presence or absence of lymph node involvement.

Only about 40% of patients are found to be operable on exploration and only 5% are alive 5 years after resection. Because of early detection in Japan the 5 years cure rate is between 80% and 90%.

Gastrointestinal stromal tumours (GIST), formerly leiomyoma.

1. Common in the stomach and small intestine. It arises from Cajal cells that lie within the stomach wall.
2. May present with abdominal mass, haematemesis or dyspepsia.
3. May reach a large size.
4. May have a pseudocapsule and recurrence is common after surgery
5. It is treated by local gastric resection.
6. Gleevac is tumour specific monoclonal antibody against some tyrosine kinases of the tumour. It has shown excellent clinical results.



Gastrostomy

A gastrostomy is an opening (stoma=mouth) of the stomach on the skin. It may be temporary or permanent.

Temporary gastrostomy

Indications

1. To decompress the bowel as an alternative to postoperative nasogastric intubation. It is more

Fig. (30.44) Stamm's gastrostomy

comfortable and does not interfere with deep respiration and coughing which are encouraged to avoid pulmonary complications. It is of advantage in the elderly with chronic pulmonary disease.

2. To feed patients who are undergoing a series of operations on the mouth and pharynx and cannot ingest food orally for weeks or months.

Technique

1. **Stamm's gastrostomy** is the traditional method (Fig. 30.44).
 - a. A small puncture is made in the middle of the anterior surface of the stomach.
 - b. A large Malecot (Mushroom head) or de Pezzar tube is inserted an inch or two into the lumen.
 - c. Two concentric pursestring silk sutures are used to invert the stomach wall, forming a snug seal, and a serosal lining to the tube tract.
 - d. The tube is brought out through a tightly fitting stab wound in the left upper quadrant.
 - e. The stomach is firmly attached to the abdominal wall by sutures around the exit site of the tube to prevent early retraction and leakage into the peritoneum.
 - f. When the gastrostomy is no more needed, the tube is simply pulled out. The track closes spontaneously because it is lined by serosa.
2. **Percutaneous endoscopic-assisted gastrostomy (PEG)** is increasingly gaining popularity (Fig. 9.2).
 - a. A gastroscope is inserted into the stomach and is used to inflate it.
 - b. The gastroscope light is made to shine through the anterior stomach wall and anterior abdominal wall.
 - c. Under local anaesthesia a trocar and cannula puncture the anterior abdominal wall and enter the stomach.
 - d. A self-retaining tube is introduced through the cannula into the stomach and its balloon is inflated.

Permanent gastrostomy

Indications

This operation is gradually losing favour as there are now better ways of dealing with advanced oesophageal carcinoma.

Technique

The aim of this operation is to fashion a track that is lined by mucosa between the stomach and the skin. The track will not close spontaneously whether a tube is left in place or not. The most popular method is the Depage-Janeway gastrostomy (Fig. 30.45).

- A full thickness rectangular flap is formed from the anterior stomach wall based on the greater curvature.
- The stomach wall defect is then closed in two layers, and the flap is converted into a tube.
- The fashioned gastric tube is passed through a stab in the abdominal wall to the left of the operation incision, and its end is sutured to the skin.

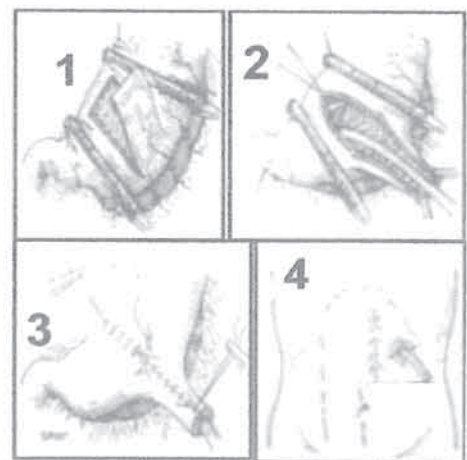


Fig. (30.45) Depage-Janeway gastrostomy

Bariatric surgery

Synonyms; Weight reduction surgery, surgery for morbid obesity.

Obesity is a chronic disease. Surgical treatment is reserved for those who have a severe degree of obesity that is called morbid obesity.

Grades of obesity

The degree of obesity can be measured by calculating the body mass index (BMI).
 $BMI = \text{Body weight in kg} / \text{Height in metres}^2$.

Ideal weight 18-25

Overweight 25-30

Mild obesity 30-35

Moderate obesity 35-40

Severe (morbid) obesity > 40

Morbid obesity responds poorly to conservative treatment and has a high incidence of complications. Bariatric surgery is the practical solution.

Complications of obesity

The higher the grades of obesity, the worse are the complications.

Physical health complications

- Hypertension
- Diabetes mellitus
- Sleep apnoea
- Coronary heart disease
- Spine degenerative diseases and disc prolapse
- Osteoarthritis of weight-bearing joints as hips and knees
- Thromboembolism
- Difficult vascular access and airway and postoperative hypoxia
- Gastro-oesophageal reflux and risk of pulmonary aspiration
- Shorter life span than lean persons

Mental health complications

- Depression

Social complications

- Inability to get certain jobs, e.g., in the military service
- Difficulties with having relationships with the opposite sex
- Difficulties with transportations and with clothing

Prerequisites for bariatric surgery

- BMI >40 or BMI >35 in the presence of obesity comorbidity, e.g., hypertension ; diabetes.
- Failure of conservative measures of weight reduction.
- Age above 18 years.
- Exclusion of endocrine causes of obesity as Cushing's syndrome hypothyroidism.
- Acceptable operative risk.
- Patient is well-informed.

Principles of bariatric operations

Bariatric surgery is done on the gastrointestinal tract. Liposuction and dermalipectomy (excision of excess skin and fat) are cosmetic operations that are intended to improve body contours but not to produce significant weight loss in the morbidly obese person.

- All bariatric operations can be done either by open surgery or by laparoscopy.
- There are two mechanisms of inducing weight loss by these operations;

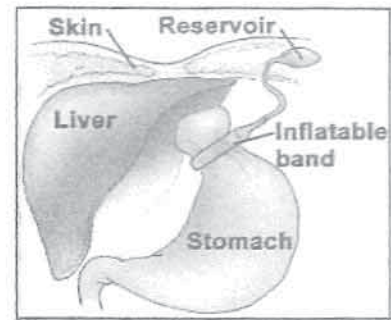


Fig. (30.46) Adjustable gastric band

- **Restricting food intake** by inducing early satiety. This is achieved by fashioning a small sized pouch from the stomach. This pouch is continuous with the esophagus. When a very small portion of food is taken, the pouch is distended and gives a sense of satiety. Food later flows down the gastrointestinal tract. Commonly-performed restrictive operations include placement of an adjustable gastric band (Fig. 30.46) and sleeve gastrectomy (Fig. 30.47).
- **Inducing malabsorption.** Part of the small intestine is bypassed so that a degree of malabsorption is induced. Commonly a bariatric operation combines malabsorption with restriction of food intake. An example is Roux en Y gastric bypass (Fig. 30.48). With malabsorptive surgery the patient should receive life-long vitamin and mineral supplements.
- Bariatric surgical procedures commonly cause loss of 55-75% of excess body weight within two years and greatly reduce the risk of obesity comorbidities.

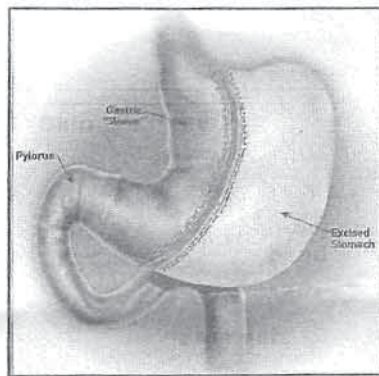


Fig. (30.47) Sleeve gastrectomy

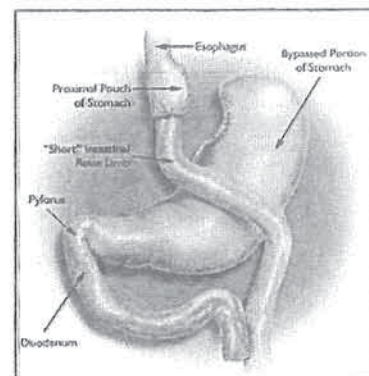


Fig. (30.48) Roux-en-Y gastric bypass

رقم الإيداع ١٧٧٩٢ / ٢٠٠٣

الترقيم الدولي 4 - 188 - 203 - 977 I.S.B.N.